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E.H.B.

TRANSACTIONS

OF THE

MEDICAL SOCIETY

OF THE

STATE OF NEW YORK

FOR THE YEAR 1903

PUBLISHED BY THE SOCIETY

1903

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MEDICAL SOCIETY OF THE STATE OF NEW YORK

ORGANIZATION

By an act of the Legislature passed in 1806 the Medical Society of the State of New York is organized as follows:

The Society is composed of Delegates and permanent and honorary members.

DELEGATES are sent by county medical societies, certain medical colleges, incorporated voluntary medical societies, and by the New York Academy of Medicine. Each county medical society is entitled to five delegates for each Assembly district in said county; incorporated medical colleges and voluntary medical societies, which are admitted to representation under provisions in the by-laws, to one delegate each; and the New York Academy of Medicine to five delegates. A delegate must be a member of a county medical society. County medical societies are by the by-laws required to pay to the Treasurer for the use of the Society five dollars annually for each Assembly district in the county, and other organizations the same for each delegate they are entitled to send; no delegate is permitted to register at the Annual Meeting until the dues of the organization he represents have been paid. Delegates are elected for a term of three years.

PERMANENT MEMBERS are elected at the Annual Meeting of the Society. Eligibility is secured by service as a delegate for three years and attendance and registry as such at not less than two meetings; membership in a county medical society is also a requisite. Permanent members pay an initiation fee of five dollars and annual dues of the same amount.

The Society may elect eminent physicians of other states or countries to *Honorary Membership* who have been nominated at a previous meeting, not to exceed two in number in any year. Physicians present at any meeting who are members of county medical societies can be made *Members by Invitation* for the meeting. These have all the privileges of the meeting except the right to vote or hold office.

EIGHT DISTRICTS, following the lines of the Judicial Districts, into which the State is divided, may each by the members present from it at an Annual Meeting elect a member of the COMMITTEE OF NOMINATION. They are as follows:

First District—New York County.

Second District—Kings, Queens, Suffolk, Richmond, Westchester, Rockland, Putnam, Orange, Dutchess Counties.

Third District—Albany, Rensselaer, Schoharie, Greene, Columbia, Ulster, Sullivan Counties.

Fourth District—St. Lawrence, Franklin, Clinton, Essex, Hamilton, Fulton, Montgomery, Schenectady, Saratoga, Warren, Washington Counties.

Fifth District—Onondaga, Oneida, Herkimer, Oswego, Lewis, Jefferson Counties.

Sixth District—Otsego, Delaware, Madison, Chenango, Cortland, Tompkins, Schuyler, Chemung, Tioga, Broome Counties.

Seventh District—Monroe, Wayne, Cayuga, Seneca, Yates, Ontario, Livingston, Steuben Counties.

Eighth District—Erie, Niagara, Orleans, Genesee, Wyoming, Allegany, Cattaraugus, Chautauqua Counties.

THE ANNUAL MEETINGS of the Society are held in the city of Albany, on the last Tuesday in January, and other meetings at such time and place as may be determined at any legal meeting. Fifteen members constitute a quorum for the transaction of business.

OFFICERS AND COMMITTEES

Elected January 29, 1903.

PRESIDENT,

A. T. BRISTOW, Brooklyn.

VICE-PRESIDENT,

EDWARD B. ANGELL, Rochester.

SECRETARY,

FREDERIC C. CURTIS, Albany.

TREASURER,

O. D. BALL, Albany.

COMMITTEE OF ARRANGEMENTS,

HERMAN BENDELL, Albany.
A. E. DAVIS, New York.
WILLIAM J. NELLIS, Albany.
HOWARD VAN RENSSELAER, Albany.
FRANCIS E. FRONCZAK, Buffalo.
ARTHUR G. ROOT, Albany.

COMMITTEE ON BY-LAWS,

H. D. WEY, Elmira.
NATHAN JACOBSON, Syracuse.
F. C. CURTIS, Albany.

COMMITTEE ON HYGIENE,

J. L. HEFFRON, Syracuse.
GEORGE B. FOWLER, New York.
M. A. VEEDER, Lyons.
JOSEPH D. CRAIG, Albany.
ELIAS H. BARTLEY, Brooklyn.
J. H. PRYOR, Buffalo.
D. V. STILL, Johnstown.

COMMITTEE ON LEGISLATION,

FRANK VAN FLEET, New York.
ARTHUR G. ROOT, Albany.
ERNEST WENDE, Buffalo.

COMMITTEE ON ETHICS,

EDWARD S. WILLARD, Watertown.
JOHN H. PRYOR, Buffalo.
LEWIS W. ROSE, Rochester.

COMMITTEE ON PRIZE ESSAYS,

A. JACOBI, New York.
J. M. MOSHER, Albany.
A. VANDER VEER, Albany.

COMMITTEE OF PUBLICATION.

F. C. CURTIS, Albany.
WILLIAM C. KRAUSS, Buffalo.
DANIEL LEWIS, New York.
O. D. BALL, Albany.

MINUTES OF THE PROCEEDINGS

OF THE

Medical Society of the State of New York

AT ITS NINETY-SEVENTH ANNUAL SESSION

HELD AT

ALBANY, JANUARY 27, 28 AND 29, 1903

FIRST DAY.—*Morning Session*

THE PRESIDENT, DR. HENRY R. HOPKINS, of Buffalo, called the meeting to order on January 27, 1903 at 9:45 A. M., in the Common Council chamber of the City Hall, Albany, and the exercises were opened with prayer by the Rev. John Rathbone Oliver, of Albany

THE PRESIDENT then delivered his

INAUGURAL ADDRESS

MR. PRESIDENT, GENTLEMEN: In addressing the Medical Society of the State of New York on the occasion of its ninety-seventh annual meeting, the speaker is not unmindful of the solemn obligations of the hour, the supreme moment of his life. I address you in grateful acknowledgement of your exceeding partiality, in confident anticipation of your forbearance and consideration for whatever comes short of your expectation.

I also offer you hearty congratulations upon the prosperous condition of our Society and of the entire medical profession throughout the State.

Our Society is soon to complete a full century of continuous strenuous, successful corporate life. During this long period we have enjoyed in a large measure the confidence of the people, and this confidence has found expression in grants of charter rights of priceless value. These charter privileges provide that the present and the future of the medical profession are in the hands of its own representatives, its chosen members, men who in their several localities enjoy the greatest measure of esteem and confidence of their fellows. What it means, that our profession with its many interests, political, sociological and scientific, should be free to develop itself, free to grow in efficiency, in influence, and in public confidence under the direction of its own members of highest ideals, of wisest judgment, time alone may unfold and measure.

Certainly we who are permitted to review the past, to mark its progress in many directions and with greater particularity its progress in improved facilities for teaching medicine, and in improved methods of such teaching—its higher and still higher standard of medical education, we, who are permitted to note that this rising, swelling tide of progress in medical education is not confined to the institutions of this State alone, but now extends to every State of our country, assuredly, we may not contemplate this, without worthy pride in the achievements of the past, and with enthusiasm and confidence in the future.

I have alluded to the many charter privileges of this Society—privileges which together constitute its birth-right, its priceless heritage. Two of these merit special mention. In the charter of the Medical Society of the State of New York, granted in 1806, and in all of the earlier amendments of that charter, the composition of the Society as determined by the number of delegates which might be sent to it from the several county societies, and other corporations entitled to representation, was cautiously, jealously and wisely guarded. Their number and length of service was determined, and from time to time changed by statutory enactment. It is my profound conviction that to this wise provision of the fathers this Society owes its century of continuity, and its well established reputation for progressive conservatism.

In the year 1880 this Society, after mature deliberation, took a forward step in gaining the confidence of the people, by repealing its Code of Ethics an instrument of many virtues but of some false philosophy and vicious tendencies, in that it purposed to deny to certain members of our profession one of their constitutional and statutory rights. The repeal in question was a virtual acknowledgment upon the part of this Society of a past error in judgment and a distinct proclamation that hereafter its customs, rules and regulations were to be distinctly loyal to the spirit and the letter of the laws of the State.

Following this important adjustment and possibly determined by it, came in 1885 a generous grant of power to the State Society in the way of authority "to regulate and control its own membership" whereby the Society was enabled to extend more than five-fold its number of delegates, to receive from the several county societies five delegates instead of one for each assembly district and to receive delegates from each of the medical colleges of the State and from voluntary medical societies at its own discretion.

This increase in charter privilege is a significant indication of growing confidence upon the part of the people in the wisdom of our Society, and constitutes an epoch in our history, a new departure in progress-making of the most auspicious potentiality.

In 1890 a still more generous gift was added to the already large number of charter rights of this Society, by the enactment of a law to provide for the examination and licensure of all candidates for the privileges of the practice of medicine in the State of New York. This law was re-enacted in 1893 and is now known as Article Eight, Practice of Medicine, Chapter 661, Public Health Law, and is probably the most considerable grant of power and privilege ever bestowed upon a medical Society in the history of the world, in that it provides for the examination by this Society through its Board of Examiners of every person desiring to practice medi-

cine in this State, even if that person is a graduate of the most exacting university of America or of Europe, or a professor therein. For a full decade we have enjoyed this remarkable privilege, this magnificent opportunity for raising the standard of the preparedness of the individuals of our profession, for the protection of the public health.

With honorable pride I congratulate you upon our long and worthy past in confident expectation that the wisdom, the loyalty and the high ideals of our members are a sure guaranty, that our future will be alike creditable.

Discoveries and improvements in pathology, diagnosis and therapeutics, have within a few years well-nigh revolutionized the practice of the art of medicine, and make an ever increasing demand upon that practice for unusual accuracy in the making of observations and records, and in the use of instruments of precision, in the surgical or medical care of the sick. This demand has been answered by the appearance of the trained nurse, a modern product, but recently a rather picturesque anomaly—but to-day a purely beneficent necessity, the doctor's reliable assistant and invaluable associate. The work of the nurse is second in importance only to that done by the physician, the emoluments are attractive and the vocation is honorable—the supply is of varied quality. Training schools for nurses now abound, and they are possibly more numerous and some are more badly equipped for their work than were our medical colleges even in our darkest days. Many hospitals maintain training schools where there are all the facilities for doing good work, in teaching and training nurses, and where sufficient time, three years, is given to a class. Many other hospitals are engaged in the same work where all these advantages are conspicuously lacking.

The requirements of the public health demand an average ability and preparedness upon the part of the nurse as upon the part of the doctor. The graduate nurses of our better training schools have recently organized and become incorporated as the New York State Nurses Association and now seek legislation "that will ultimately place Training Schools for nurses under the supervision of the University of the State of New York." I have received through our Secretary a copy of the proposed bill, and careful study of the document convinces me that the movement is wise, timely, in good hands, and deserves the hearty support of this Society. I, therefore, advise that the matter be referred to the Committee on Legislation with instructions to support the measure in every way possible.

The question of the prevention of consumption continues to be one of the few great topics of the time. I note, and with pleasure report, that throughout the world more and more attention is devoted to this subject and that too by earth's wisest and most powerful persons. From all sides and in all quarters prominence is given to three thoughts, (1) the prevention of consumption is the most important responsibility of the day and the world; (2) The prevention of consumption is the practical method of dealing with the scourge; and, (3) The prevention of consumption is the first duty of the State. These great principles have for years guided this Society in its action upon this subject, and it is with unusual pleasure that I report briefly that after long and discouraging delay, definite progress is now being made in the work of establishing a State sanatorium for cases of incipient consumption. A suitable site of some 500 acres has been selected and purchased and a building to give accommodations to 100

patients is under contract. I hope and trust that this Society will continue to give to this important question its warmest sympathy, and most active co-operation, to the end that our people be kept informed of the nature of their danger from this preventible disease, and that the full power of the State be put and continued in operation for the prevention of tuberculosis and the saving thereby of our people from the enormous losses in time, money, sickness and death, from its ravages.

Drifts and tendencies in medical practice like currents and tides of ocean are to be carefully noted, some for avoidance, others for following. For many years medical practice has shown a distinct inclination to produce specialists and of the work of our specialists it may be observed that some of it is fussy and lacking in art, some of it is pretentious and too highly priced, but in the main it is in the direction of accuracy, efficiency and progress. Special training, leading to unusual preparedness is the dominant note of the times and is as appropriate to medicine as to other forms of personal or corporate activity. The scientific discoveries and the progress of the last half century have established upon permanent foundations that most important department of medicine, the science of hygiene. State medicine, one of its important subdivisions, has won and now holds for our profession the respect and confidence of the wise of this world to a degree not hitherto attained. It is in every way to our advantage that this respect and confidence should be preserved and fostered. The work of State medicine seems to be a proper field for the activities of the specialist in hygiene and the degree of Doctor of Public Health or its equivalent to be reached after special preparation would suitably attest the holder's readiness for the work. For years the universities of Great Britain have given courses leading to this degree, and by law of the realm the degree of D. P. H. is required of candidates for certain official positions.

It is recommended to your earnest consideration, that this Society invite the State Medical Societies of 1857 and 1865 to unite with it in an appropriate memorial to the honorable legislature of the State, praying that authority be granted to the Regents of the University to fix the requirements for, and power to grant the degree of Doctor of Public Health or its equivalent.

It would seem that this step may properly be taken at once and that further legislation, in case such should be needed, indicating the privileges of the holders of such degrees, might properly await future developments.

Your attention is directed to the significant fact that this is the ninety-seventh annual meeting of our Society and that subsequent to this but two annual meetings will occur before we enter upon our second century of corporate life. The century has witnessed a remarkably well sustained growth in confidence and trust upon the part of the legislature towards the organized medical profession and medical institutions, a confidence which has borne rich results in increasing favors, powers, and privileges, granted at different times, to meet existing needs. No member of the medical profession can review the statutes which to-day constitute the charter of our Society and its constituent county societies without just pride in his profession which has been the recipient of so many testimonials of confidence and honor at the hands of the sovereign people. It would seem in many ways desirable that we observe our approaching centennial in a manner

in some degree becoming so notable an occasion. Such events furnish opportunities for review of progress, increase respect and stimulate proper ambition, attract the attention and arouse the interest of possibly lukewarm members of our profession, and also the notice of the general public not alone to what has been so creditably accomplished, but also to the many important and desirable objects of our future plans and purposes.

Ample time, constructive thought and our wisest and best efforts may well be devoted to this matter to the end that the observances may meet the rightful requirements of the occasion.

Should this matter meet your approval, and should it be deemed expedient at this time to take initiatory steps, it is advised that the chair be authorized to appoint a committee to consider the matter of how we may appropriately recognize the coming centennial of the Medical Society of the State of New York with instructions to report its conclusions to the annual meeting in 1904.

The relation of the medical profession to the authority of the State has for more than a century been much in the thought of the wisest of our members. Of these relations it may be observed that they have always been intimate and important, though sometimes disturbed by misunderstandings. From 1806 to 1857 the profession was a unit, having a distinct organization in the State, and in its several counties. This organization is fashioned after that of our country with distinct powers and privileges reserved to the county societies, the analogues of the States, and with higher and more general powers enjoyed by the State Society, the head of the medical State, as Congress is the head of the general government. The relation of the several independent corporations, county and State is by statutory provision, and is a wise adjustment of the principles of local independence and home rule, coexistent and co-operative with general government and direction by a central body, representative in character, of delegate composition, acting for the entire profession under statutory direction and limitations. This organization after the style and manner of our country is believed to be best for the general purposes of human government, and well adapted to the more specific purposes of medical societies the diffusion of true science, and particularly the knowledge of the healing art.

In 1857 the unity of the medical organization of the State was broken by the creation of a second system of societies, State and county, having the same general and local powers, and privileges as those authorized in 1806, and in 1865 was still further interrupted by the authorization of a third system of medical societies State and local; and since that date there has existed in our State the monstrous anomaly of three distinct classes of physicians having equal authority to practice medicine, and equal privilege to enjoy the honors and emoluments of that practice; that this unfortunate condition of medical malorganization was inimical to the best interests of our profession history will abundantly attest. It is for us to appreciate the significance of the fact, that the responsibility for these unfortunate and deplorable divisions, was, and is, ours. There were differing views as to the manner in which the art of medicine should be practiced, and the majority were not tolerant and considerate of the rights of the min-

ority, and drove that minority to the Legislature for protection. To heal these wounds, to compose these differences, to reunite the medical profession of this State upon the broad, Catholic, liberal basis of the medical law of 1806, is one of the first duties of this Society, the Society of the dominant and responsible majority.

Fortunately, it is my high privilege to congratulate you upon the fact, that this Society after mature deliberation, after full consideration and discussion, resolutely undertook this important duty and for more than twenty years has striven in the direction of a restoration of legal medical unity to the medical profession of the State upon the basis of the medical law of 1806.

Your attention is invited to two important steps taken in this cause, the repeal of the Code of Ethics, then known as the Code of the American Medical Association; and the campaign before the Legislature for the State examination and license, with the physicians of 1857 and 1865 as companions and allies, these are now matters of history, and also important records of progress.

A third, and it is hoped a final campaign in this cause was inaugurated by my predecessor of last year, in the appointment of a committee upon "The Relations of the Medical Society of the State of New York to the National Profession." As an incident of our efforts to restore legal medical unity in this State, this Society has been without representation in the American Medical Association for many years. As was pointed out in the inaugural address of my immediate predecessor "because of the encouragement received by the reorganization upon a broad platform of the American Medical Association" it seemed desirable that we make an overture in the direction of a resumption of diplomatic relations with the national body. The progress of the work you will hear in the report of the committee. The untimely death of Dr. Abel Mix Phelps created a vacancy in the committee which vacancy I filled by the appointment of Dr. Frank Van Fleet of New York. I trust my action in this appointment will meet with your approval.

The unseemly and undignified divisions of the medical profession of our State are not a credit to the commonwealth or the profession and are distinctly out of harmony with the spirit of the century. I have the conviction that it were well, if this whole matter, primary, and incidental, should be adjusted before this Society completes its present century, that it is more important that when the question is adjusted and settled, it shall be settled right, with all due consideration on the part of the majority for the rights and privileges of the individuals who constitute the minority; but this Society can never with honor agree to any adjustment that sacrifices the principles for which we have so long contended, and which have led to the placing the State of New York in the front rank, not only in this country but throughout the world in the matter of proper requirements for admission to the practice of medicine.

I now declare the ninety-seventh annual meeting of the Medical Society of the State of New York open for the transaction of business.

On motion the following committee was appointed to consider the recommendations contained in the Address: D. B. St. John Roosa, of New York; W. S. Ely, of Rochester; W. C. Krauss, of Buffalo.

THE PRESIDENT announced the following committees:

Business Committee: Ernest Wende, Buffalo; Hamilton D. Wey, Elmira; J. Montgomery Mosher, Albany.

Committee on Credentials: B. U. Steenberg, Albany; Judson P. Pendleton, Brooklyn; A. W. Van Slyke, Coxsackie.

Committee on Nominating Candidates for Vacancies on the State Board of Medical Examiners: John O. Roe, Rochester; James D. Spencer, Watertown; Daniel Lewis, New York; Herman Bendell, Albany; Henry L. Elsner, Syracuse.

The last named committee subsequently reported the following for these vacancies: William Warren Potter, Buffalo; William S. Ely, Rochester; M. J. Lewi, Albany; W. J. Nellis, Albany; Hamilton D. Wey, Elmira; Edward B. Angell, Rochester. This report was accepted and adopted.

Reports of officers and Standing Committees were presented. [See Reports.]

The Committee of Arrangements presented a brief verbal report through its chairman, Dr. Herman Bendell, Albany.

THE TREASURER presented his report, which was referred to an Auditing Committee: L. Duncan Bulkley, New York; William Maddren, Brooklyn; James D. Spencer, Watertown. This committee subsequently reported that it found the report correct.

DR. NATHAN JACOBSON, Syracuse, presented the report of the Committee on By-Laws. Report accepted.

DR. JOHN L. HEFFRON, Syracuse, presented the report of the Committee on Hygiene. Report accepted.

DR. GEORGE B. FOWLER, New York, introduced the following resolution, which was adopted:

Whereas, Strenuous efforts are being made to induce the Legislature to utterly undo the great advances made in recent years in tenement-house reform and sanitation, and to prevent the permanent carrying into effect of the great improvements in the construction and management of tenement-houses in New York and Buffalo, which has been made possible by the recent Tenement House Law; and

Whereas, Pulmonary tuberculosis is practically epidemic in the City of New York, there being 8,000 deaths and 20,000 cases annually; and

Whereas, The lack of light and air in the rooms of the great masses of the people is one of the most potent causes in increasing the prevalence of this and other communicable diseases and constitute serious menaces to health and morality,

Be it Resolved, That the Medical Society of the State of New York deplores any weakening of the present Tenement-House Laws, and urgently requests the Legislature to permit no changes to be made that will in any way decrease the amount of light and air available for the people living in such houses, or in any way take a backward step in regard to their sanitary conditions.

DR. ARTHUR G. ROOT, Albany, presented the report of the Committee on Legislation. Report accepted.

DR. A. T. BRISTOW, Brooklyn, presented the report of the Committee on Ethics.

THE PRESIDENT ruled that the different matters considered in this report be taken up in the order in which they were reported.

On motion of Dr. A. T. Bristow, the petition of the Watertown Society was granted.

On motion of Dr. D. B. St. John Roosa, the recommendation of the com-

mittee in regard to the former members of the Lewis County Medical Society was unanimously adopted.

DR. A. JACOBI, New York, for the Committee on Prize Essay, stated that this committee had no report to make.

DR. F. C. CURTIS, Albany, presented the report of the Committee on Publication. Report accepted.

On motion of Dr. D. B. St. John Roosa, seconded by Dr. Frank Van Fleet it was directed that the *Transactions* be sent to Delegates from County Societies.

DR. WILLIAM WARREN POTTER, Buffalo, presented an abstract of the Annual Report of the State Board of Medical Examiners. Report accepted.

THE SECRETARY presented an abstract of the Reports of the Committees of the Eight Judicial Districts, appointed at the last Annual Meeting, on Relations with County Medical Societies. The purpose of their appointment was to act in concert with officers of the County Medical Societies in their respective districts, to assist in all matters relating to the growth, social activity and scientific work of these Societies. (See pp. 12, 20 and 22, *Transactions* of 1902.) They were requested to report as to whether there is a demand in the County Societies for more liberal representation; whether the recent change increasing the number of delegates affected the attendance of delegates on the Annnal Meeting of the State Society; how generally are physicians in the State members of their County Societies.

Reports received from all of the District Committees indicate that in the more urban counties a more liberal representation is desired while in the rural counties it is not, although upon this opinions vary. The Societies in many of the counties are in a flourishing condition, most of the physicians are active members and their meetings are well sustained. Counties not having active Societies were reported; in the Third District, one; in the Sixth, one; in the Eighth, two, whilst two others failed to co-operate with the committee in correspondence or otherwise. Much was done by all of the committees in not only communicating with the County Societies but in personal attendance upon their meetings and in furnishing papers before them.

THE secretary added to the summary of these reports data from his records showing that there were registered in attendance upon the last Annual Meeting, at which the increase in the number of delegates first became operative, 306 delegates from County Medical Societies, the number at the meeting of 1901 having been but 100. Of the sixty-one counties there were representatives from all but sixteen, of which number eight have been regularly represented in recent years; four, viz.: Genesee, Saratoga, Schuyler and Wyoming have had no representation since 1884; Ontario, Orleans and Wayne, only a few times; and the Putnam County Society has ceased to exist, whilst the Rockland County Society recently revived is flourishing and well represented at this meeting. Further, of permanent members there were 131 registered at the last meeting. Of 200 members now living who became members prior to 1889, about one-half are still on the active list of the Society; and of 462 who have become members since 1889, 351 are on the active list, forty-two have died, seven resigned, and four become non-resident. Report received.

DR. HENRY L. ELSNER, Syracuse, presented the report of the Committee on Relations of the Medical Society of the State of New York to the Medical Profession. [See Reports.]

On motion the report was received, directed to be placed on file and sent to the members of all County Medical Societies.

The Business Committee then took up the program of the meeting.

DR. HERMAN C. GORDINIER, Troy, read a paper, "Arguments for the Existence of a Separate Cortical Centre for Writing."

On motion a recess was taken at 12:45 P. M.

FIRST DAY.—*Afternoon Session*

The Society reconvened, the Vice-President, D. William A. Moore, of Binghamton, in the chair.

The Committee of Nomination was announced as follows:

Appointed by the President.—William Warren Potter, of Buffalo.
First District—A. E. Davis, of New York.
Second District.—William Maddren, of Brooklyn.
Third District.—A. Vander Veer, of Albany.
Fourth District.—R. J. Wilding, of Malone.
Fifth District.—Nathan Jacobson, of Syracuse.
Sixth District.—Arthur W. Booth, of Elmira.
Seventh District.—William M. Brown, of Rochester.
Eighth District.—Arthur E. Hurd, of Buffalo.

DR. HENRIETTA P. JOHNSON, New York, read a paper, "Medical School Inspection in the City of New York."

DR. CHARLES G. WAGNER, Binghamton, read a paper, "The Care of the Insane."

DR. FLOYD S. CREGO, Buffalo, read a paper, "Differential Diagnosis of the Familiar Forms of Spinal Disease."

The foregoing papers were discussed by Dr. F. S. Crego, of Buffalo, and Dr. Wagner made some closing remarks.

DR. REYNOLD W. WILCOX, New York, read a paper, "Erythroploeum; a Clinical Study."

DR. JAMES K. CROOK, New York, read a paper, "American Climates Suitable for Phthisical Patients."

Discussion by Dr. Alfred Meyer, of New York, with closing remarks by Dr. Crook.

DR. D. H. WIESNER, New York, read a paper, "Retinoscopy."

DR. FRANCIS VALK, New York, read a paper, "The Physician and the Ophthalmoscope."

DR. SAMUEL M. BRICKNER, New York, read a paper, "Incomplete Transverse Congenital Occlusion of the Vagina."

DR. PETER H. BRYCE, Secretary of the Provincial Board of Health of Ontario, read a paper, "Some Scientific and Practical Details Regarding Vaccine and Vaccination."

On motion of Dr. M. J. Lewi a vote of thanks was tendered Dr. Bryce for his instructive paper, and the latter was referred to the Committee on Publication.

DR. EDGAR S. THOMSON, New York, read a paper, "Treatment of Purulent Conjunctivitis."

DR. JUSTIN L. BARNES, New York, read a paper, "Transportation and the Ophthalmic Referee."

Discussion by Dr. Percy Fridenberg, of New York.

DR. LUCIEN HOWE, Buffalo, read a paper, "Eye Strain and Headache."

Discussion by Dr. Francis Valk, of New York, and closing remarks by Dr. Howe.

FIRST DAY.—*Evening Session*

The Society reconvened at 8:30 P. M., at the call of the President.

DR. HENRY D. INGRAHAM, Buffalo, read a paper, "Hydrops Tubae Profluens."

DR. C. W. M. BROWN, Elmira, read a paper, "Sterilized Milk, Pasteurized Milk or Clean Milk."

DR. HENRY L. K. SHAW, Albany, read a paper, "The Examination of Milk by the General Practitioner."

Discussion by Dr. Walter A. Scott, of Niagara Falls, and Dr. E. E. Smith, of New York.

The next order was a Symposium on Haematology.

DR. IRVING P. LYON, Buffalo, read a paper, "Blood Examination in General Practice."

DR. THOMAS R. BROWN, of Baltimore, Md., being unavoidably detained, his paper, "The Eosinophiles, their Etiology and Value in Diagnosis and Prognosis," was read by Dr. L. Duncan Bulkley.

DR. J. C. DACOSTA, JR., of Philadelphia, Pa., read a paper, "Degeneration of the Erythrocyte."

DR. EDWIN ALLEN LOCKE, of Boston, Mass., read a paper, "The Iodine Reaction and its Diagnostic Significance."

On motion of Dr. H. L. Elsner, of Syracuse, the thanks of the Society were extended to the invited guests who had presented this interesting series of papers.

SECOND DAY.—*Morning Session*

The Society was called to order by the President at 9:45 A. M.

THE SECRETARY read the following list of applicants for permanent membership, recommended for membership by the Committee of Nomination, and moved their election; viz.: Frederick H. Brewer, Utica; William B. Brinsmade, Brooklyn; Luzerne Coville, Ithaca; John H. Doughty, Matteawan; George Bingham Fowler, New York; William B. Hanbidge, Ogdensburgh; William Henry Happel, Albany; William E. Hayes, Frankfort; James Zalmon Hunt, Lowville; John Francis Hunter, Mamaroneck; Edward Marshall Hyland, Utica; Thomas Z. Jones, Waterville; Jack Killen, Binghamton; Sherwood Le Fevre, Amsterdam; W. Clinton Kellogg, Syracuse; Thomas Masson, Cape Vincent; Sylvester James McNamara, Brooklyn; James H. Mitchell, Cohoes; George Stephen Munson, Albany; Leo Handel Neuman, Albany; Daisy M. Orleman, Peekskill; Charles Winfield Pilgrim, Poughkeepsie; Ezra B. Potter, Rochester; Frank Walker Sears, Binghamton; Eugene Alfred Smith, Buffalo; Thomas P. Scully, Rome; Wilbur D. Wolff, Rochester; William Henry Zabriskie, Glen Cove.

They were duly elected on compliance with the By-Laws.

DR. JOHN L. HEFFRON, of Syracuse, offered the following resolutions, which were adopted:

Resolved, That in view of the inadequate provision for the care of the consumptive poor of the State of New York, the Medical Society of the State of New York endorses the efforts of the State for the establishment by it of a Hospital for incipient cases of tuberculosis;

Furthermore, That it deplores the insufficiency of the appropriation for and the delay in opening the institution at Ray Brook, three years having elapsed since the initial appropriation; and

Furthermore, That it would urge upon the Governor and Legislature adequate moral and financial support of the Board of Trustees of this institution in order that their work may redound to the credit of the State of New York; and

Furthermore, That a copy of these resolutions be forwarded to the Governor and all State officers and to all members of the Legislature, duly signed by the President and Secretary of the Medical Society of the State of New York.

DR. ROBERT H. M. DAWBARN, New York, read a paper, "The Surgeon's Enemy, the Skin."

Discussion by Dr. H. D. Ingraham, of Buffalo.

DR. RAMON GUITERAS, New York, read a paper, "The Technique of Prostatectomy."

DR. WILLIAM S. BAINBRIDGE, New York, read a paper, "Periduodenal Abscess Secondary to Perforative Ulcer of the Duodenum."

Discussion by Dr. L. S. Pilcher, of Brooklyn, and H. L. Elsner, of Syracuse, the discussion being closed by Dr. Bainbridge.

DR. FREDERIC R. STURGIS, New York, read a paper, "Some Points Regarding the Treatment of the Functional Disorders of the Sexual Organs in the Male."

Discussion by Dr. R. H. M. Dawbarn and A. Jacobi, of New York, with closing remarks by Dr. Sturgis.

DR. CHARLES A. L. REED, of Cincinnati, Ohio, delivered a "Memorial Address on the Life and Work of Professor Rudolph Virchow."

DR. A. JACOBI, of New York, made some supplementary remarks on Virchow, and then moved a vote of thanks to Dr. Reed for his address. The motion was carried unanimously.

DR. WILLIAM A. MOORE, the Vice-President, having taken the chair, Dr. Henry R. Hopkins of Buffalo, delivered the President's Address. At its conclusion the thanks of the Society were extended to him.

SECOND DAY.—*Afternoon Session*

The Society reconvened at 2:45 P. M., and in the temporary absence of the President and Vice-President, Dr. H. L. Elsner took the chair.

DR. W. S. ELY, of Rochester, presented the report of the Committee on the President's Inaugural Address:

Your Committee to consider the recommendations in the President's Inaugural Address would respectfully report as follows:

First: We recommend that the bill for the "Registration of Nurses of

New York State'' be referred to the Committee on Legislation, with instructions to urge its passage in its present form, or with such modifications as may seem to said Committee advisable.

Second: We recommend that this Society expresses its interest in the prevention and cure of tuberculosis, and will actively co-operate in all just measures having these ends in view.

Third: We consider timely, so much of the President's Address as refers to the special education and training of physicians for sanitary work, and recommend such action by this Society as may result in legislation giving to the Regents of the university authority to establish the requirements for, and power to grant the Degree of Doctor of Public Health.

Fourth: Your Committee is impressed with the profound significance attaching to the 100th meeting of this Society to be held in 1907. Such an event should be celebrated in a way commensurate with its importance, and to this end we suggest that a committee of members, distinguished for long identification with, knowledge of, and active interest in this Society be appointed by the chair, to arrange a program for our Centennial Meeting, and to outline the same at the next Annual Meeting of this Society.

All of which is respectfully submitted.

D. B. St. John Roosa,
William S. Ely,
William C. Krauss,
Committee.

On motion of Dr. Frank Van Fleet, of New York, the report was received, and the recommendations contained in it adopted.

Dr. Van Fleet further moved that the Medical Society of the State of New York hold a semi-annual meeting in the County of New York some time in October, 1903. Seconded by Dr. A. Jacobi and carried unanimously.

Dr. Harvey R. Gaylord, Buffalo, read a paper, ''Plasmodiophora Brassicae.''

Dr. Arthur W. Elting, Albany, read a paper, ''Primary Carcinoma of the Vermiform Appendix.''

Discussion by Dr. Willis G. Macdonald, of Albany.

The next order was a Symposium on Arterio-sclerosis.

Dr. Delancey Rochester, Buffalo, read a paper, ''The Early Recognition and Symptoms of Arterio-sclerosis.''

Dr. Glentworth R. Butler, Brooklyn, read a paper, ''Arterio-sclerosis and the Heart.''

Dr. Irving P. Lyon, Buffalo, read a paper, ''Arterio-sclerosis and the Kidney.''

Dr. Charles G. Stockton, Buffalo, read a paper, ''Arterio-sclerosis and the Digestive System.''

Dr. William Browning, Brooklyn, read a paper, ''Arterio-sclerosis and the Nervous System.''

Dr. Adolf Meyer, New York, read a paper, ''Arterio-sclerosis and Mental Disease.''

Dr. Thomas E. Satterthwaite, New York, read a paper, ''Pulsus Infrequens.''

The general discussion was opened by Dr. E. Libman, of New York, and was continued by Dr. A. L. Benedict, of Buffalo, Dr. Nelson G. Richmond, of Fredonia, Dr. Edward D. Fisher, of New York, Dr. H. L. Elsner, of Syracuse, Dr. Anna M. Stuart, of Elmira, and Dr. James J. Walsh, of New York. Closing remarks were made by Drs. Rochester, Stockton and Satterthwaite.

THE PRESIDENT announced the appointment of the following as the Committee on the Centennial Anniversary of the Society: Dr. D. B. St. John Roosa, of New York, Chairman; Dr. A. Jacobi, of New York; Dr. A. Vander Veer, of Albany; Dr. Daniel Lewis, First District; Dr. George R. Fowler, Second District; Dr. Willis G. Macdonald, Third District; Dr. Charles Stover, Fourth District; Dr. Henry L. Elsner, Fifth District; Dr. Hamilton D. Wey, Sixth District; Dr. William S. Ely, Seventh District and Dr. William Warren Potter, Eighth District.

DR. HEINRICH STERN, New York, read a paper, "The Management of the Uraemic State."

Discussion by Dr. Thomas H. Manley, of New York.

DR. A. L. BENEDICT, Buffalo, read a paper, "Hepatic Ballottement or Bimanual Palpation."

THIRD DAY.—*Morning Session*

The meeting was called to order by the President at 10 A. M.

DR. THOMAS P. SCULLY, Rome, read a paper, "Cancer of the Cervix; Treatment by the X-Ray; Report of Cases."

Discussion by Dr. F. C. Curtis, of Albany, and closing remarks by Dr. Scully.

DR. A. H. TRAVER, of Albany, read a paper, "An Operation for Cicatricial Contractures of Upper Extremities."

DR. S. B. WARD, of Albany, moved a vote of thanks to the Business Committee, the Committee of Arrangements and the Committee on Credentials for their very satisfactory performance of their respective duties. Carried.

DR. J. M. MOSHER moved that the Secretary be authorized to place the list of the American Association of Medical Libraries, with its modifications as they occur, upon his mailing list, so that the Transactions of the Medical Society of the State of New York may be distributed to the libraries of this Association. Seconded.

THE SECRETARY replied that if the libraries referred to were established in their position the motion was unnecessary, for the by-laws direct that the Transactions shall be sent to such. He had made an effort to complete the files of libraries in this respect.

The motion was then carried, the Chairman ruling that it was in the nature of an instruction to the Secretary to enlarge the list of medical libraries receiving the Transactions.

THE SECRETARY moved that the President-elect be authorized to appoint a committee, consisting of two from each judicial district, with the Vice-President-elect as the Chairman of the committee, to co-operate with the local societies in their efforts to broaden the influence of the State Society. Carried unanimously.

DR. ARTHUR W. BOOTH presented the report of the Committee of Nomination:

The Committee of Nomination makes the following report:

For President: Hamilton D. Wey, of Elmira, (6 votes); A. T. Bristow, of Brooklyn, (2 votes).

For Vice-President: Edward B. Angell, Rochester.

For Secretary: Frederic C. Curtis, Albany.

For Treasurer: O. D. Ball, Albany.

For Committee of Arrangements: Herman Bendell, Albany; Arthur G. Root, Albany; W. J. Nellis, Albany; Howard Van Rensselaer, Albany; A. E. Davis, New York; Francis E. Fronczak, Buffalo.

For Committee on By-laws: Hamilton D. Wey, Elmira; Nathan Jacobson, Syracuse; F. C. Curtis, Albany.

For Committee on Hygiene: J. L. Heffron, Syracuse; George B. Fowler, New York; M. A. Veeder, Lyons; Joseph D. Craig, Albany; Elias H. Bartley, Brooklyn; J. H. Prior, Buffalo; D. V. Still, Johnstown.

For Committee on Legislation: Frank Van Fleet, New York; Arthur G. Root, Albany; Ernest Wende, Buffalo.

For Committee on Ethics: A. T. Bristow, Brooklyn; Edward B. Angell, Rochester; Edward S. Willard, Watertown.

For Committee on Prize Essays: A. Jacobi, New York; J. M. Mosher, Albany; A. Vander Veer, Albany.

For Committee of Publication: F. C. Curtis, Albany; Daniel Lewis, New York; William C. Krauss, Buffalo; O. D. Ball, Albany.

For Delegates to the Fourteenth International Congress of Medicine, at Madrid, Spain: Ramon Guiteras, New York; Harvey R. Gaylord, Buffalo.

For Delegates to the Ontario Medical Society: T. H. Halstead, Syracuse; W. M. Brown, Rochester.

For Delegate to the Connecticut State Medical Society: L. Duncan Bulkley, New York.

For Delegates to the New Jersey State Medical Society: W. H. Haskin, New York; Edgar A. Vander Veer, Albany.

For Delegate to the Massachusetts State Medical Society: C. H. Richardson, Albany.

For Delegates to the Canadian Medical Association: George A. Madill, Ogdensburgh; A. L. Benedict, Buffalo.

For Honorary Membership: Francis X. Dercum, of Philadelphia, Pa.; R. H. Chittenden, of New Haven, Conn.

For Eligibility to Honorary Membership: Peter H. Bryce, of Toronto, Canada.

Respectfully submitted,

WM. WARREN POTTER,
Chairman.

ARTHUR W. BOOTH,
Secretary.

On motion of Dr. A. Jacobi, of New York, the report was received.

THE PRESIDENT called attention to the fact that the by-laws required that when the Committee of Nomination is unable to act unanimously in the nomination of an officer, it must present the two names of candidates receiving the highest number of votes. He then declared that the order of business was balloting for the office of President.

DR. HAMILTON D. WEY, of Elmira, said: I should feel insensible if I failed to appreciate the honor conveyed in this nomination, but I think that logically and geographically the presidency this year should go to Kings county. I, therefore, ask unanimous consent for permission to withdraw my name.

On motion of Dr. Jacobi, consent was given to Dr. Wey to withdraw his name, with expression of appreciation of his reasons and motives, and Dr. A. T. Bristow, of Brooklyn, was thereupon duly elected President of the Society.

The other officers as nominated, were duly elected, and the rest of the report of the committee adopted, the President-elect being authorized to fill the vacancies made in the Committee on Ethics by the election of part of its membership to office.

The following papers were announced as read by title:

"Memorial of Edward M. Moore," by William S. Ely; "Memorial of Abel Mix Phelps," by Frank Van Fleet; "A Contribution to the Surgery of the Bile Duct," by John J. McGrath; "Operative Possibilities in Cases of Advanced Carcinoma of the Breast," by Lewis S. Pilcher; "Acute Osteomyelitis," by Arthur W. Booth; "Diagnosis in Cholelithiasis, with Illustrated Cases," by Willis G. Macdonald; "Injuries and Infections of New-born Children," by Irving M. Snow; "Improvements, Attained and Prospective, in the Care of the Insane," by Frederick Peterson; "Prevention of Puerperal Eclampsia," by John T. Wheeler; and "On the Pathology, Diagnosis and Treatment of Malignant Growths," by Leonard Weber.

DR. HENRY R. HOPKINS: The business of the ninety-seventh meeting of the Medical Society of the State of New York has been concluded. It but remains to me to thank you for the very great honor which you have conferred upon me in permitting me to preside over a meeting of your great Society. It is also my privilege to congratulate you upon the remarkable evidences which come from every quarter of our great State, that the love and loyalty and enthusiasm of the medical profession for this great Society are growing and strengthening, and are full of the highest promise for our future. I thank you for your exceeding courtesy.

DR. S. B. WARD: I am sure every one of us appreciate the zeal and hard work given by the retiring president during the past year, and I move that the thanks of the Society be tendered to Dr. Hopkins for his admirable conduct of his office. Seconded and carried unanimously.

DR. HOPKINS: I thank you, Mr. President and gentlemen. I now declare the ninety-seventh session of the Medical Society of the State of New York to be concluded.

The Society adjourned.

There were registered in attendance at this meeting 250 Delegates and 140 Permanent Members.

FREDERIC C. CURTIS,
Secretary.

The next Annual Meeting of the Society will be held at Albany, January 26, 27 and 28, 1904.

REPORTS

1. Treasurer's Report

The Medical Society of the State of New York in account with the Treasurer.

Balance in Treasury January 30, 1902	$2990 66
Received from County and other affiliated Societies	840 00
Received from permanent members, dues	2040 00
Received from sale of *Transactions*	181 00
Interest accrued on deposits in Savings Bank	100 13
Total	$6151 79

Expenditures from January 30, 1902, to January 27, 1903.

Secretary, services and expenses of office	$389 50
Treasurer, services and expenses of office	192 50
Committee on Legislation, expenses of	232 00
Executive Committee, expenses of, (New York meeting)	122 50
Business Committee, expenses of	13 61
Transactions, printing of	1076 06
Transactions, expressage of	125 32
Annual Meeting: Stenographer, messengers, annual dinner,	435 00
Stationery and printing	247 10
Binding	11 00
Judge Andrews, legal opinion	50 00
Insurance and rent of storage loft	49 25
Overpaid dues refunded	10 00
Total	$2953 84
Balance in Treasury January 27, 1903	$3197 95

2. Report of Merritt H. Cash Fund

Interest accrued to date of last report		$237 82
Interest accrued during 1902		24 28
		$262 10
Expenditures:		
Paid Dr. Wm. C. Krauss	$1 50	
Paid Tiffany & Co.	100 00	
		101 50
Balance, interest account		$160 60

The Fund ($500) and interest accrued are in deposit in the Albany Savings Bank.

O. D. Ball,
Treasurer.

3. Report of the Committee of Publication

To the Medical Society of the State of New York:

The volume of *Transactions* for 1902 consisted of an edition of 850 copies, containing 527 pages, and costing $1076. Estimates were secured for the work which was given to the Brandow Printing Company, of Albany, as the lowest bidder.

The Committee of Publication had placed before it the printing of the material of two meetings, 40 papers having been presented at the semi-

annual meeting and 50 at the annual. Of these 62 found a place in the volume, besides the Anniversary Address of the President and the Prize Essay. Certain papers of unusual length and technical composition were not introduced. By closer printing, abbreviation, and omitting the usual lists of members of County Medical Societies the essential material of the two meetings was incorporated into the volume without exceeding the customary size, to which it seems wise to the Committee to adhere. Containing with other good matter the Symposium papers on three vital topics which are exhaustively treated, together with illustrations to embellish it, and of typographical excellence it will fill a worthy place in the series which comprise the annual production and record of the Society.

There were sent to members about 350 copies, to exchanges, libraries and complimentary 130, sold to individuals 85, to County Medical Societies 100, leaving nearly 200 copies on hand. This excessive remainder is due to the small number sold both to individuals and to County Societies. As to the former we have again provided for the subscription to the next volume by individual members of the profession at one dollar a copy although the result of effort in this direction has not been very encouraging; its intrinsic value as a collation of fresh material, much of it from very competent writers, ought to secure a wider circulation. The County Medical Societies comparatively few of them, are taking quotas of it and it is not likely that more than the number thus disposed of this year will be exceeded.

These *Transactions* have formed a series now for nearly 100 years that constitute a permanent record of the work of the Society and indeed of the profession during that long period, and as such it appears that they should be maintained as the best way of continuing it. It does not seem however that a volume prepared at such cost and labor, although distributed to our permanent members and deposited in all public medical libraries in this and other states, should be limited in its distribution to so small a number. It is a question which we might propose to the Society whether it would not be judicious to distribute it likewise gratuitously to the delegate members of recorded attendance upon the meetings, limiting the number given, to the number of Assembly Districts in their county. This would widen its circle of distribution by probably 300 copies, and it would not add perhaps more than $100 to the additional cost of the edition.

F. C. CURTIS,
WM. C. KRAUSS,
DANIEL LEWIS,
O. D. BALL,
Committee.

4. REPORT OF THE COMMITTEE ON LEGISLATION

To the Medical Society of the State of New York:

The policy of the Legislative Committee, during the last session of the Legislature, has been more defensive than offensive. There was a marked diminution in the number of bills having any bearing upon medical affairs. The medical bills introduced during the session of 1902 numbered only seventy-eight. Of these nineteen became laws; forty-four died in committee, and did not reach the Governor; eight were unsigned by the Governor, and seven were vetoed by the mayors of cities. The larger proportion of these bills were purely local measures.

The effect of the recent outbreak of smallpox was shown in the introduction of several bills for permission to create contagious hospitals. Two bills were introduced by the anti-vaccinationists, calling for a commission to investigate the value of vaccination, but neither were reported. A compulsory vaccination bill was introduced by Senator McCabe but did not come to a vote. An amended vaccination bill, giving extra power to the State Commissioner of Health, also died in committee.

The bill of greatest interest to the medical profession at large was one

introduced by Senator Brackett entitled "An act to regulate the practice of osteopathy." The limitations of osteopathy were sufficiently discussed in the last report of your committee, and the subject need not be dwelt upon at this time. This bill was actively opposed and arrangements made for a public hearing at the time when the State Medical Society was in session. This hearing was largely attended by medical men, and some able arguments were advanced against the bill. The result was most gratifying, as the Senate judiciary committee voted, by a large majority, not to report the bill. This bill was ingeniously drafted and was similar to the present medical law, providing for a State Board of Osteopathic Examiners. To be eligible for examination candidates must have been graduated from a regularly-conducted school of osteopathy, and have personally attended at least four terms of five months each. That is to say, a man could study osteopathy for twenty months, pass the Osteopathic Board of Examiners, and be entitled to all the privileges afforded a regular practitioner, who is obliged to spend at least four calendar years in a medical college. The majority of persons, unfortunately, are not able to judge for themselves as to what constitutes fitness in a physician, and in the interests of public health this bill was defeated.

A particularly vicious measure was introduced relieving hospitals from liability in case of negligence on the part of its employes and others. If a hospital had no responsibility in the case of people in its employ, or attached to it, it would not use so much care in its choice of nurses and physicians. The results would be disastrous to the patients' best good, and would make suits for damages against doctors and nurses more numerous.

A bill amending the insanity law excited much discussion and a widespread interest. This was an administration measure and finally became a law.

The only legislation urged by the Legislative Committee was a bill introduced as a result of conference between the State Board of Regents and the State Medical Society. It was called "an act to amend the public health law relative to admission and examination in certain medical studies." It allows as the equivalent of the first year (of the four years' prescribed medical course) evidence of graduation from a registered college course. This bill encourages men to take a college degree before entering upon the study of medicine, and will thereby raise the standard of medical students. The Regents can also admit applicants nineteen years of age to a preliminary examination in anatomy, physiology, hygiene and chemistry, after two years' study, at which time they have completed the course in the primary branches.

It is with much pleasure that your committee at this time, takes advantage of the opportunity to express to Dr. H. L. K. Shaw our most sincere thanks. Much of the success that has attended the work of your committee we feel was due to his faithful, conscientious efforts as Secretary of the Legislative Committee.

Your committee takes pleasure in saying that its efforts have, at all times, been along lines sanctioned by the State Board of Regents, and upon those designed to protect the public by a proper standard of medical fitness.

In the chief executive, Governor Odell, we have always found an ardent supporter of educational standards, and we feel that so long as the State Society continues its present course the future of higher educational requirements in medicine is secure.

FRANK VAN FLEET,
ARTHUR G. ROOT,
ERNEST WENDE,
Committee.

5. Report of the Committee on By-Laws

To the Medical Society of the State of New York:

The Committee on By-laws respectfully reports as follows:

1. Examination and approval of the by-laws of the Watertown Academy of Medicine.

2. The Medical Society of the County of Kings submitted a copy of its constitution and by-laws. The latter contained a clause which was opposed to and at variance with the by-laws and sentiments of this Society. Your committee so advised said Society.

3. The by-laws in force at the present time retain much of old phraseology, as is apparent by comparison with those embraced in the printed issues under date of 1854, 1880, 1890, and 1897.

The most important amendments and changes that have occurred in the by-laws during the last twenty-one years may be briefly summarized as follows: in 1882 it was declared "The System of Medical Ethics adopted by the Society February 7th, shall be considered authoritative to govern the medical profession in the State of New York." This "System of Medical Ethics" superseded the Code of Ethics of the American Medical Association, and remained in force till 1893 when it was abolished by a vote of the Society. In 1894 it was provided that candidates for vacancies in the State Board of Medical Examiners shall be appointed by a committee of five, appointed by the President of the Society, their selection to be reported to the Society for its approval before being forwarded to the Board of Regents. It is in addition provided, in case of emergency making it necessary for the Board of Regents to request nominations from this Society between the annual meetings, this committee is instructed to report directly to the Board of Regents. In 1895 that section of the by-laws providing for the election of Censors to attend final examinations in medical schools was annulled. At this time membership in a county society was declared a qualification for delegateship to the State Society. The time of meeting was changed from the first Tuesday in February to the last Tuesday in January. The conditions upon which incorporated voluntary medical societies should have representation was defined in 1896; in 1900 two years existence was demanded instead of three years as formerly. In 1896 three years was prescribed as the term of office of delegates and an actual attendance upon two meetings. The rearranging the Districts of the State for the election of members of the Committee on Nominations was suggested in 1898 by the adoption of the present Judicial Districts, eight in number, "in the place of the obsolete Senatorial Districts of like number," and the same incorporated in the by-laws of 1900. In the last named year county societies were granted increased representation—five delegates from each Assembly District.

We believe the increased representation of county societies, and recognition of incorporated voluntary medical societies, will bring the above named societies in closer touch than formerly with the State or parent Society, causing the latter to become a more representative delegate body in closer touch and harmony with its affiliated bodies.

The legal status of the Medical Society of the State of New York remains unchanged. The Society enjoys to-day the same rights, privileges and prestige as was conferred in the past by various legislative enactments. In whatever way it might have been affected by adverse legislation and the repealing of laws relating to and governing it, by virtue of the Revised Statutes, 1901, Vol. II, p. 2287, it still maintains its place and dignity as the Medical Society of the State of New York, endeared to its members through its connection with the past, its adherence to its convictions, and what it represents for all that is good and best in medicine.

Hamilton D. Wey,
Nathan Jacobson,
F. C. Curtis,
Committee.

6. Report of the Committee on Ethics

To the Medical Society of the State of New York:

1. October, 1902, application was received from the Medical Society of the City of Watertown for admission to representation in this Society. It being found that all the requirements of the by-laws are met, it is recommended that the application be granted.

2. Charges were presented at the last meeting of this Society by the Medical Society of the County of Lewis against two members of this Society, Drs. A. N. Crosby and C. E. Douglas, of Lowville, of unprofessional conduct, in that they were reported to have instigated a suit for malpractice against Dr. Wm. H. Johnson of Port Lyden, and others, and asking that they be dropped from membership. This matter on reference to your committee has been investigated by inquiry of both parties to the case. The evidence to which weight was attached was that furnished by the official minutes of the trial of Dr. Johnson, a copy of which is filed with this report, the weight of which compels the committee to report favoring the petition of the Lewis County Society.

Respectfully submitted,

A. T. Bristow, *Chairman.*
E. B. Angell,
E. S. Willard,
Committee.

7. Report of the Committee on Hygiene

To the Medical Society of the State of New York:

Your Committee on Hygiene has studied the various problems, the solution of which is of importance to the entire State, and presents the following report:

1. *Tuberculosis* continues to be the most prevalent of the infectious and preventable diseases, notwithstanding each successive year sees a slight diminution of the death rate from this scourge. The fact that the imperfect and inadequate methods thus far available for the limitation of this disease have given such results, should arouse the enthusiasm of every physician and every philanthropist, and should unite them in most vigorous efforts to push forward all well known and necessary measures for stopping the spread of this disease. In England during the last fifty years the diminution of deaths from tuberculosis has been forty-five per cent. Dr. Hermann M. Biggs says in a recent article: "During the last twenty years there has been a reduction in the death rate from tuberculosis diseases in New York City of nearly forty per cent." These figures indicate how responsive to even slightly improved conditions this disease is. If we place side by side with this improvement in a whole nation and in the greatest city in the United States, the figures printed by Bowditch, showing that in the Massachusetts State Sanatorium at Rutland seveny-nine per cent. of the cases received in the first stage were "arrested," we can say boldly that of all infectious and communicable diseases tuberculosis is the most easily controlled. But the percentage of improvement leaves an appalling number who are yet victims of this disease. Biggs recently said that an expert diagnostician could doubtless find tuberculosis in 30,000 people in New York City to-day. In a recent publication it was shown that in New Hampshire, from 1884 to 1901, 40.72 per cent. of all deaths between the ages of twenty and thirty were due to tuberculosis. It is because of this, and because this disease strikes down men and women in the most productive period of their lives, that your committee wishes to emphasize our duty to this largest class of unfortunates. We, therefore, recommend that persistent agitation of the duty of the community to the tubercular poor be infused with new enthusiasm. (*a*) The education of all the people in the fundamental principles of infection and of self-protection

should be brought about through the general adoption of such measures as are in force in New York City. To reach the largest number of the people, industrial insurance companies and all sick benefit societies could be made use of for the distribution of literature with great promise. The senior students in every grammar school and high school should be required to pass an examination in the method of preventing the spread of tuberculosis. Societies for the Prevention of Tuberculosis should be organized in every community. Public lectures in each school district should be frequently given by one who understands the facts and has the gift of teaching them to minds of ordinary intelligence. By these educative efforts much could be done to teach the teachable the sanitary science of every-day living. The degenerates and irresponsible will have to be attended to directly by the administrative authorities, and compelled to conform their lives to methods that are not dangerous to their neighbors. (*b*) We note with alarm the efforts said to be making by the greedy to secure a repeal of the Tenement House Act, or to so modify it as to make it practically inoperative. Much of the improvement in the management of tuberculosis in the past has been due to a slowly attained improvement in the housing of the poorer classes. The argument that such a restrictive law for any but overcrowded sections is unnecessary is specious and fallacious. On the other hand, it is right that the most stringent regulations governing the building of tenements should be made to apply to all cities large enough to contain a single tenement or flat house. We, therefore, recommend that the influence of this great Society be powerfully exerted to preserve all the good of the present law and to increase its humane provisions as rapidly as possible. (*c*) We recommend the compulsory registration in the proper office by every physician of every case of tuberculosis, and that in case of death the house be thoroughly disinfected by the proper health officers. (*d*) We recommend the opening of dispensaries for the treatment of the tuberculous poor, and that the attending physicians be instructed to seek out those whom they may learn are showing signs of beginning tuberculosis, though they may yet be at work. (*e*) We recommend the establishment in every city, or at the county seat, in parts of the State not thickly inhabited, a Receiving Hospital for Tuberculosis. This hospital should be erected by each community and be independent of the County Poor House. To it should be sent every case of tuberculosis among the tubercular poor, and these cases should be sent at the earliest possible period of their disease. The cases here received should be under competent observation, and receive the most scientific treatment for a sufficient time to determine an accurate prognosis, when from them should be selected the most favorable cases, which should be sent to the State Sanatorium for Tubercular Poor, while those in whom the prognosis is less favorable be still given the scientific treatment. Such favorable cases need not all be incipient, for nothing is surer than that cases in almost any stage of the disease are curable under certain well ascertained conditions. Finally, we urge the State to prosecute, with great diligence, the building of the one hospital, for which an appropriation of $150,000 has been secured. On December 31, 1902, we had the pleasure of observing that the cellar had been commenced. When this hospital shall have been finished and put under the right management, and when it shall have been properly conducted for a single year, we predict that such results will have been attained that a legislature which willingly appropriates almost four million dollars annually for the housing and care of her insane and epileptics alone, unfortunates who seldom become self-supporting, though some of them may be discharged, will not fail to see that that larger class of more easily cured patients and more menacing to the community, the tuberculous poor, are cared for in a way as intelligent and effective. Then legislators may have an object lesson which will illustrate the estimate of the Charity Organization Society, which is to the effect that "there is a total annual loss to the city (New York) from tubercular diseases of the poor of $23,000,000."

2. *Typhoid Fever* has not claimed as many victims in the past year as in years previous. This is apparently due to a diminished influx of infected

individuals rather than to any marked improvement in the care of water supplies. Attention has been effectively drawn to other carriers than water of the infectious principle in a paper by Dr. M. A. Veeder, of our Committee, which has attracted the attention of all sanitarians. In Circular No. 62 from the Adjutant General's office, on "Typhoid Contagion and the Means of Preventing It," issued December 26, 1902, most careful orders have been issued to all army posts which, if carried out, will make impossible such another sacrifice of the lives of the soldiers from this disease as disgraced our army in the Spanish-American war. In a paper read before the Syracuse Academy of Medicine, Dr. R. H. Hutchings, Acting Superintendent of the State Hospital at Ogdensburg, traced an epidemic of typhoid in that institution to infected ice cut in a bay of the St. Lawrence River, and he demonstrated that the bacilli, after imprisonment in ice for seven months, were active and capable of cultivation on culture media. This brilliant demonstration has established the fact that the bacillus typhosus is viable and virulent after imprisonment in ice for a period much longer than has ever before been regarded as possible. It has been reported that the waters of the larger lakes in the Adirondacks have become polluted with typhoid dejecta, and attention is called to this as a new source of danger. In view of these facts your committee recommends that it be made imperative that all dejecta from typhoid patients be thoroughly disinfected before they are disposed of in any way whatsoever.

3. *Smallpox* has gradually increased during the past ten years, and during the last year has been more prevalent than ever. Through the opposition of unscientific sentimentalists persistently kept up in several widely circulated journals, valuable for some of their literary and artistic features, and through the revolt of many people against subjecting themselves to septic wounds, which have been the result of unscientific methods of slovenly physicians, the value of vaccination has been more and more called into question, until in many communities a generation of unvaccinated people has grown up. If the world has not been convinced of the efficacy of vaccination against smallpox, it cannot be convinced of the truthfulness of any scientific deduction, nor of any mathematical demonstration. Argument will no longer avail and is undesirable. In a report of the Health Officer of Rochester to this committee it is apparent that the epidemic in that city during the past six months has had for its victims only those who have never been vaccinated, or those in whom the immunity conferred has run out in the changes attending the establishment of puberty. The exceptions to this rule are so very few in the history of all recent epidemics everywhere that they but accentuate the general truth of the proposition. To have an epidemic of smallpox in the first years of the twentieth century is a disgrace to our boasted civilization and is an unpleasant commentary on the work of physicians. Your committee would direct attention to the necessity of strengthening the existing laws making vaccination compulsory in childhood, and again after the establishment of puberty, and urge the conscientious enforcement of this law by the health officers of every community.

4. *Scarlet Fever* has been endemic and epidemic in all portions of our State for many years. The cases have hitherto been light, and there has been a laxity in carrying out the provisions of quarantine. Your committee has received the report of an epidemic of virulent scarlet fever in Amsterdam, in which city there have been forty deaths from this disease in the last three months. Recent cases in Albany and Syracuse have assumed a virulent form. This serves to call attention to the possibility of a change in the virulence of this disease throughout the State as has been observed many times in the history of this and other epidemic diseases. We, therefore, recommend that unusual watchfulness be observed by health officers and attending physicians, and that a quarantine for at least six weeks be strictly maintained, until every manifestation of the disease has ceased, however light it may appear to be.

5. *Cancer.*—The increasing prevalence of cancer and the present unsatisfactory state of our knowledge concerning the etiology of this disease

cause us to recommend that the State appropriation for the laboratory investigation of this disease be continued.

6. *Impure Milk.*—The problem of how to obtain a supply of wholesome milk for large cities is still one of the most serious proportions. The natural difficulties of obtaining from a hairy animal kept under artificial conditions of life a secretion that will be free from injurious elements are and always must be very great. Fortunately, we believe, the latest dictum of Koch, that bovine tuberculosis is not transferable to man has made no impression upon the laws regulating the production of milk. With the accumulated evidence against his proposition, it is clear that we must insist upon the rigorous enforcement of all our laws made for stamping out bovine tuberculosis. So long as there is a shadow of a question of the accuracy of his deductions it would be criminal for us to jeopardize the life of a single infant by permitting the use of milk from a tuberculous cow. In the London Congress of Tuberculosis it was shown that while the general death rate in England from tubercular diseases had in fifty years diminished forty-five per cent. the death rate from abdominal tuberculosis in infants had increased twenty-seven per cent. and that no attention had been given to improving the condition of the milch cows. The success of the Medical Society of the County of New York in improving the milk supply by certifying the milk of approved dairies has been attended with such remarkably good results that your committee recommends that the subject be taken up in every County Society and in all societies affiliating with this organization and that each carry out the plan now successfully operated by the Society of the County of New York, in collaboration with Boards of Health.

7. *Incompetency of Health Officers.*—Finally, your committee feels that the present imperfect condition of Sanitary Science in our country is largely due to the fact that health officers are too frequently appointed for political reasons rather than because of their peculiar fitness for the positions which they occupy. In almost no medical school of our State or in the country is there more than the most superficial teaching in State Medicine, and no incentive is held out to men entering the medical profession to devote their lives to this most important field of labor. We may well take lesson from some of our older sister nations, and now, when we are just becoming recognized as a world power in finance and diplomacy, we may well consider whether it is not right that we should elevate the plane of preventive medicine. The contributions of American medical men have not been inconsiderable. The achievements of the Medical Department of the Army in banishing yellow fever and cholera from our insular possessions should but be added incentive to the performance of great work in all departments of State medicine. Since 1866 England has had a law making it necessary for every man occupying the position of health officer to a community to have a special Diploma in Public Health, granted upon examination by one of several recognized schools, in subjects covering the whole field of State medicine. Your committee, therefore, asks you to call the attention of the Regents of the State University to this matter and to ask them to carefully consider this subject and provide for the examination of all physicians candidates for the position of health officers in the State, and to grant to the successful candidates a special Diploma in Public Health. And to the end that this may operate for the permanent welfare of the whole State, it asks that you request our Committee on Legislation to frame such laws as in their judgment seem necessary to make it imperative upon all communities in the State that have health officers to require the proof of especial fitness for the duties of the office to which they aspire.

Respectfully submitted,

JNO. L. HEFFRON,
GEO. B. FOWLER,
JOSEPH D. CRAIG,
D. V. STILL,
Committee.

8. Report of the State Board of Medical Examiners

To the Medical Society of the State of New York:

I am instructed by the State Board of Medical Examiners representing the Medical Society of the State of New York to present to you an abstract of its annual report for the academic year as follows:

Total number of candidates for license since the establishment of the boards, 7034; successful, 5528 or 78.5 per cent.; unsuccessful, 1506 or 21.4 per cent.; total number of candidates from July 31, 1901, to Aug. 1, 1902, 928, of these there applied for full license 685, of which number 558 were successful. Of the above number 601 took the full examination at one time. 84 of the number had previously passed in the primaries and were examined only in the so-called practical branches. Of the above number appearing before this board, 17.8 per cent. were rejected; of those appearing before the Homœopathic board, 13.2 per cent. were rejected; of those appearing before the Eclectic board, 50 per cent. were rejected. The above candidates for license appeared as follows: Before this board 610, successful 501; before the homœopathic board 53, successful 46; before the eclectic board 22, successful 11.

Candidates for the primary examinations, viz., in anatomy, physiology and hygiene and chemistry, who had studied these subjects for two years, appeared as follows: Before this board 318, successful 314 or 99 per cent.; before the homœopathic board 35, successful 35 or 100 per cent.; before the eclectic board 5, successful 5 or 100 per cent.

The average rejections in each of the seven topics was 47+.

The minimum rejections for the year, 34, were in obstetrics; the maximum rejections for the year, 57, were in pathology and diagnosis.

The number of candidates for medical license in this State has been decreasing since 1898, at which time the maximum, 869 was reached. This year there were but 685.

During the past eleven years the average per year appearing before the State board was 586+; before the homœopathic board 46+; before the eclectic board 16+.

The advanced standards of New York State medical schools are evidently appreciated as manifested by the increasing number of undergraduate medical students in this State, 2563; an increase of 248 over last year. The postgraduate students number 1072, an increase of 42 over last year.

The State boards of examiners should not be compared with examining boards of the army, navy and hospital marine service. They serve different purposes; we to declare credentials sufficient, they to differentiate between those already declared competent.

The board advocates (under proper supervision and by legislative action) the additional postgraduate degree of D. P. H. (doctor of public health).

Shall allowance for experience be made in examination of older practitioners coming from other states and countries, exempting them from the primary branches? The board believes that some measure should be adopted to make less exacting the test for such practitioners.

The reasons for non-ability to reciprocate with sister states is explained because of the lower standards of these latter.

Written medical examination tests are not all that could be desired, but as administered by the Regents they are far preferable to any system of bedside examination whereby the identity of the candidate is necessarily revealed.

Experience with cases now under advisement should indicate a proper rule of procedure in the recognition of credentials for admission to advanced standing in medical schools on the part of graduates of academic schools. We advise liberal treatment of these cases till such rule is formulated.

The board will again strive to have the present arrangement of topics altered to conform with methods of teaching.

The following were elected officers for the succeeding year:
President: William Warren Potter, M.D.
Secretary: Maurice J. Lewi, M.D.
Question Committee: George Ryerson Fowler, M.D., and Maurice J. Lewi, M.D.

I am also directed to state that the report proper of the board, which after a decade of active experience treats of various phases of medical education and of medical interest generally gleaned from that experience, constitutes too lengthy a document to read at this time, and to request that this body accept the abstract of the report as here presented, with the understanding that a copy of the full report will be mailed to each member and delegate of the Society as soon as printed.

Respectfully submitted,

WILLIAM WARREN POTTER, *President.*
WM. S. ELY,
A. WALTER SUITER,
GEO. RYERSON FOWLER,
JOSEPH P. CREVELING,
EUGENE BEACH,
MAURICE J. LEWI, *Secretary.*

REPORT OF THE COMMITTEE OF CONFERENCE

Submitted to the Society at its Annual Meeting, January, 1903

At the last meeting of the Medical Society of the State of New York your president, in his Inaugural Address, after considering the reciprocal relations of the State and County Societies and the Relations of the Medical Society of the State of New York to the National Profession, concluded as follows: "A thorough study of this subject leads me to the conclusion that the unification of the regular profession is demanded, not only by the profession, but by the thinking public and that a reorganization can readily be brought about if reason shall prevail. *This must be accomplished without the loss of identity or individuality of this time-honored society, with dignity and without the sacrifice of principle.*" He insisted that the time had come for a final effort, which shall have for its object the gathering of the profession of this State under a single banner, upon a liberal platform, and with representation in the American Medical Association.

His recommendations seemed justified because of the encouragement received by the reorganization upon a broad platform of the American Medical Association; because it has become evident that the national body appreciates the injustice done to the Medical Society of the State of New York by its excommunication during so many years; because of a growing desire for amalgamation by individual and influential members of the profession of this State, representing over thirteen thousand consistent and conscientious workers, who fail to find any underlying principle of sufficient importance to justify the existing division; because of the influence which would be gained in the settlement of vital questions affecting the public welfare; and, finally, because of the undignified and hopeless position of a noble profession divided against itself without reason and justice.

It was recommended that the Medical Society of the State of New York appoint a committee of five to confer with an equal number representing the New York State Medical Association for the purpose of formulating a plan which shall have for its object the reorganization of the regular profession of this State, in a body in affiliation with the American Medical Association; the committee to report the result of its labors at the next meeting of the Medical Society of the State of New York.

"In the event of failure of the New York State Medical Association to appoint such a committee, or if the committees should fail to agree

upon a plan of reorganization, the committee appointed by the Medical Society of the State of New York shall have full power, if it deems it expedient, to represent this Society before the American Medical Association, and the Secretary of this body shall, if the majority of the committee desires, provide the individual members with credentials of delegates to the American Medical Association. The method of election or appointment of the committee, representing this Society, shall be decided upon by the committee to which the President's Inaugural Address shall be referred, and shall be ratified, as are all recommendations, by a vote of the Society."

The committee on the President's Inaugural Address gave these recommendations its unqualified approval and suggested that the gentleman from whom it emanated be the Chairman of the Committee and empowered to associate with himself four representative members of this Society.

The Society having by vote approved the recommendations, the President became Chairman of the Committee of Conference and appointed as his associates Drs. Abraham Jacobi of New York City, A. Vander Veer of Albany, A. M. Phelps of New York City, and George Ryerson Fowler of Brooklyn. The untimely death of Dr. Phelps was mourned by the Committee. Dr. Phelps entered upon the deliberations with great earnestness showing a desire to do all in his power to bring about the union of our State profession. He was present at two of the meetings held in New York and took an active part at these joint conferences. The Chairman of your Committee appointed Dr. Frank Van Fleet as Dr. Phelps' successor and since the death of the latter, Dr. Van Fleet has been active on the Committee.

On the 5th of February, 1902, Dr. F. C. Curtis, Secretary of the Medical Society of the State of New York, sent the following communication to the President of the New York State Medical Association:

ALBANY, N. Y., Feb. 5, 1902.

DR. ALVIN A. HUBBELL,

President of the New York State Medical Association, Buffalo, N. Y.

DEAR SIR—At a recent meeting of the Medical Society of the State of New York, held in Albany January 28th to 30th., the following recommendation contained in the inaugural address of the then president Dr. Henry L. Elsner of Syracuse, and indorsed by the Committee upon Recommendations in the Inaugural Address, was adopted:

"That the Medical Society of the State of New York appoint a committee of five to confer with an equal number representing the New York State Medical Association for the purpose of formulating a plan which shall have for its object the reorganization of the regular medical profession of this State, which body shall be in affiliation with the American Medical Association, and that the committee report the result of its labors at the next meeting of the Medical Society of the State of New York."

I trust that the purport of this action of our Society is clear, and that it may meet with a response from the Association of which you are president. I am, dear sir,

Yours very respectfully,
(Signed) F. C. CURTIS,
Secretary.

To which a supplement was added, dated,—

ALBANY, N. Y., Feb. 7, 1902.

DR. A. A. HUBBELL,

President of the New York State Medical Association, Buffalo, N. Y.

DEAR DOCTOR—I would add to my letter of the 5th inst., reporting to you the action of the Medical Society of the State of New York in proposing to appoint a committee of conference with one to be appointed by the Association of which you are president, that the following committee

has been appointed: Dr. Henry L. Elsner of Syracuse, Dr. A. Jacobi of New York, Dr. A. Vander Veer, of Albany, Dr. A. M. Phelps of New York, Dr. George Ryerson Fowler of Brooklyn.

Yours very truly,

(Signed) F. C. CURTIS,
Secretary.

At the Council meeting of the New York State Medical Association held on February 7th, 1902, the following, introduced by Dr. Ferguson, was adopted:

WHEREAS, The Medical Society of the State of New York appointed a committee to confer with a similar committee from the New York State Medical Association, with the view to a union of the two organizations, and notice of such creation of a committee having been officially given to our president, together with the request that a corresponding committee be appointed by us; therefore be it

RESOLVED, That this Council (being the Executive Board of the Association) appoint for the purpose of conference in question a committee of five, consisting of Dr. E. Eliot Harris, as chairman, and Drs. William H. Biggam, Emil Mayer, Parker Syms and Frederick Holme Wiggin, to which committee the president is added as a member ex-officio. Seconded by Dr. Gouley, and carried unanimously.

The following letter was received by Dr. Curtis, bearing date of Feb. 8th, 1902.

DR. FREDERIC C. CURTIS,
Secretary.

DEAR DOCTOR—The action of the Medical Society of the State of New York, as set forth in your communication of the 5th inst., has been referred by me to the Council of the New York State Medical Association, and it has appointed the following Committee of Conference: E. Eliot Harris, Chairman, 33 West 93rd St., New York City; Frederick Holme Wiggin, 55 West 36th St., New York City; Emil Mayer, 25 East 77th St., New York City; Parker Syms, 50 West 47th St., New York City; William H. Biggam, 1197 Dean St., Brooklyn, N. Y.

You will kindly inform your Committee of this appointment and state that our Committee is now ready to act in accordance with the purposes proposed, and may be addressed through the Chairman, Dr. E. Eliot Harris.

Yours of the 7th., announcing the committee of the State Society, is received and I have sent the names to Dr. Harris.

Yours truly,

(Signed) ALVIN A. HUBBELL,
President New York State Medical Association.

Upon receipt of this letter Dr. Curtis informed the Chairman of your Committee of the appointment of a committee by the New York State Medical Association, whereupon, the Chairman wrote the following letter to Dr. E. Eliot Harris, Chairman of the Association Committee:

SYRACUSE, N. Y., Feb. 2, 1902.

DR. E. ELIOT HARRIS,
33 West 93rd St., New York City.

MY DEAR DOCTOR—Dr. Curtis, the Secretary of the Medical Society of the State of New York, advises me of your appointment as chairman of a committee to represent the State Association at a conference with a committee composed of State Society men.

As I have the honor to be chairman of that committee, I thought it would be wise to write to you concerning the time of our meeting. Beginning on the 13th of March our Medical College closes for twelve days,

during which time it would be convenient for me to give the required time for this work.

I note that your entire committee is composed of New York men, and as a majority of our men live either in New York or near that city, the meetings in all probability had better be held there.

Will you kindly let me know at your earliest convenience whether the dates included above would be agreeable to you and to the others of your committee?

With many kind regards, I am,

Sincerely yours,
(Signed) HENRY L. ELSNER.

In answer the following letter was received:

February 24th, 1902.

To DR. HENRY L. ELSNER,
Chairman Committee of Conference,
Medical Society of the State of New York.

MY DEAR DOCTOR—Your letter of the 21st of February was this day received. Our Committee had already considered favorably the question that all communications between the two committees should be in writing, addressed to the respective chairmen, and that each committee could meet by itself to discuss all subjects pertaining to the work in hand, and its written views sent to the chairman of the other committee.

It seems to me that this manner of proceeding will not only be time-saving, but will surely be much more desirable in every other particular than by the transactions of these affairs in joint session.

Therefore permit me to suggest that your committee, through you, send to me the propositions which our committee is to consider.

Yours very respectfully,
(Signed) E. ELIOT HARRIS,
Chairman.

After consultation with all the members of the Committee of Conference, representing the Medical Society of the State of New York, your Chairman sent the following reply:

SYRACUSE, N. Y., March 5th, 1902.

DR. E. ELIOT HARRIS,
Chairman Committee of Conference,
Medical Association, State of New York.

MY DEAR DOCTOR—In reply to your letter, bearing date of February 24th, addressed to me as Chairman of the Committee of Conference of the Medical Society of the State of New York, in which you make the statement that your "Committee had already considered favorably the question that all communications between the two societies should be in writing" and that each committee should "meet by itself to discuss all subjects pertaining to the work in hand, and that its written views" should be "sent to the chairman of the other committee," and in which you lead the committee of the Medical Society of the State of New York to conclude that there shall be no conference, but simply correspondence between the two committees through the respective chairmen, I would say that the committee representing the Medical Society of the State of New York was appointed *to confer, not to correspond,* with a similar committee representing the New York State Medical Association. The committee which I represent feels that the methods of deliberation can only be settled by a joint meeting of both committees in conference, where both sides may be permitted to express themselves freely, and where each side may take into consideration the views of the other, and where both, prompted by a liberal spirit, shall be willing to reach such conclusions as may result from such

deliberation. Under no other conditions can the purposes for which we were appointed be accomplished, and the committee which I represent must refuse to act in any other way.

Awaiting an early reply, I am, for the Committee,

Yours very respectfully,
(Signed) HENRY L. ELSNER,
Chairman.

To which the following answer was received:

March 8, 1902.

HENRY L. ELSNER, M.D.,
Chairman of the Conference Committee of the Medical Society of the State of New York.

MY DEAR DOCTOR—I have the honor to acknowledge receipt of your letter of March 5th, in which you say "that the committee representing the Medical Society of the State of New York was appointed to confer and not to correspond, and the committee which I represent must refuse to act in any other way."

While the committee of the New York State Medical Association believes the method of proceeding already suggested by this committee will not only be time-saving, but will surely be desirable in every other particular, nevertheless this committee, in the interest of a united profession in one State medical body, will be glad to meet the committee of the Medical Society of the State of New York at such time and place as may be agreed upon by the two chairmen.

I am, for the committee,

Yours very respectfully,
(Signed) E. ELIOT HARRIS,
Chairman.

The chairmen of the respective committees arranged for a joint meeting in the city of New York on March 19, 1902. At this meeting Dr. Henry L. Elsner was made chairman of the joint conference. It was understood by both committees that each was acting without power and that all questions must finally be referred to the respective State bodies for ultimate action and ratification. All the members of both committees were present and the following proposition of the Committee of Conference from the New York State Medical Association to the Committee of Conference of the Medical Society of the State of New York for the union of the two bodies was presented:

Proposition of the Committee of Conference of the New York State Medical Association to the Committee of Conference of the Medical Society of the State of New York, for union of the two State bodies, presented March 19, 1902.

Two years ago, the New York State Medical Association, founded in 1884, was reorganized under a charter granted by the Legislature. Its plan of reorganization is based upon those of several other State medical associations, and has been regarded by the Committee on Reorganization of the American Medical Association as a proper basis for the organization of the American medical profession in the different states.

The Medical Society of the State of New York, formed under a law of 1806, was changed materially in 1813, after which nearly all the important privileges granted have been repealed by many subsequent acts of the Legislature, so that the basis of its existence is, to-day, so involved as to be little understood.

Therefore, in the spirit of meeting what we believe to be an honest desire to unite the regular medical profession in this State, we propose that the New York State Medical Association and the Medical Society of the State of New York be reconstituted, by an act of the Legislature,

into a State medical body to be known as the New York State Medical Society, of which all members in good standing in both bodies shall be charter members. The reconstituted State medical body shall be the representative in this State of the American Medical Association, by virtue of acceptance of the constitution and by-laws of the American Medical Association.

(Signed) E. ELIOT HARRIS, *Chairman,*
WILLIAM H. BIGGAM,
EMIL MAYER,
PARKER SYMS,
FREDERICK HOLME WIGGIN.

To which the Committee answered as follows:

The Committee of the Medical Society of the State of New York acknowledges the receipt of the communication from the Committee of Conference of the New York State Medical Association and begs to reply as follows:

Proposition of the Committee of Conference of the Medical Society of the State of New York to the Committee of Conference of the New York State Medical Association for union of the two State bodies, presented March 19, 1902.

In the spirit of meeting what we believe to be an honest desire to unite the regular medical profession in this State, we propose that the New York State Medical Association and the Medical Society of the State of New York, be reorganized by legal union into a single State medical body, to be known as the Medical Society of the State of New York of which all members in good standing in both bodies shall be charter members. The reconstituted State medical body shall be the representative in this State of the American Medical Association by virtue of acceptance of the Constitution and By-laws of the American Medical Association, adopted in 1901, except Chapter XV.

(Signed) HENRY L. ELSNER, *Chairman.*
A. JACOBI,
A. VANDER VEER,
A. M. PHELPS,
G. R. FOWLER.

It will be noted that the Committee representing the Medical Society of the State of New York made use of the words, "legal union," believing that whenever the two societies unite their interests and become a single body, such unification must of necessity follow by *legal means* and in accordance with the laws of the State of New York which laws your committee believed they would be unable to interpret without the advice of counsel. In reply to this communication the committee representing the New York State Medical Association, submitted the following in writing:

To the Committee of Conference of the Medical Society of the State of New York, acknowledging the receipt of and in response to its reply to the proposition submitted by the Committee of Conference of the New York State Medical Association.

The plan of organization of the New York State Medical Association being acceptable to your committee, we will recommend that the reorganized State medical body be known as the Medical Society of the State of New York, and the term legal union be understood to mean applying to the Legislature for a new charter.

(Signed) E. ELIOT HARRIS, *Chairman,*
WILLIAM H. BIGGAM,
EMIL MAYER,
PARKER SYMS,
FREDERICK HOLME WIGGIN.

It will be noted that the Committee of the Association believed that "legal union" necessitated application for a new charter. Upon this point, the Committee representing the Medical Society of the State of New York had taken no legal advice. However, your Committee insisted that under no circumstances would any action be taken which would for one moment interrupt the existence of the Medical Society of the State of New York.

Your Committee in its answer to the Association Committee at the second session of our joint conference on March 19th, 1902, added the words, "Except Chapter XV," because it was held by the Association Committee that Chapter XV of the by-laws of the American Medical Association included the original code of medical ethics. Chapter XV of the by-laws of the American Medical Association published in 1901 reads as follows: "These By-laws shall be in effect and force after the close of the annual meeting of 1901, provided, that the sections shall elect delegates during the session for 1901; and provided further, that nothing in these By-laws shall be construed to repeal the rules of the Association governing the relation of members to each other and to the Association."

The third session of both committees was held in the city of New York, at the Academy of Medicine April 18, 1902, at three P. M. There were present Drs. Harris, Biggam, Mayer, Syms and Wiggin, representing the Association and all the members of the Committee representing the State Society. At this meeting it was still held by the Committee representing the New York State Medical Association that in order to make *legal union* possible, the reorganized Society must of necessity apply to the Legislature for a new charter. The Association Committee at this time and at all other times since its appointment has impressed the Committee representing the State Society as being in earnest and equally anxious to bring about the union of the profession of this State, under one banner and at this time assured our Committee that in the event of reorganization, the reorganized society, including the Association members, would celebrate our one hundredth anniversary in 1906 in a manner befitting a body with a history such as ours and a record to which all members in the reorganized society might point with just pride.

At this meeting methods of reorganization were considered and a method of reorganization was suggested which had for its basis the reorganization of the regular profession of this State in the Medical Society of the State of New York, which would entitle every member of a County Society to membership in the State Society and in the American Medical Association. At this meeting was also considered the advisability of divorcing the business and ministerial affairs of the State Society from the scientific and referring these to a governing body, very much after the method now followed by the American Medical Association. At both of these meetings the individual members of the Committee representing the Medical Society of the State of New York agreed among themselves and in joint session that it would not be advisable for the Medical Society of the State of New York to send delegates to the meeting of the American Medical Association to be held in June, 1902. The minutes of the meetings were signed by the respective chairmen of the Committees as an attest of the correctness of the report of the proceedings as they related to the principal matters discussed, without binding either society in any way.

Your Committee realizing the great responsibility reposed upon it, deemed it expedient to take legal advice that it might learn from a recognized authority what measures were necessary to bring about *legal union,* and for that purpose your chairman consulted the Hon. Charles Andrews Ex-Chief Judge of the Court of Appeals of the State of New York and adds to this report the opinion of this learned counsel. It is as follows:

The following memorandum has been made on consultation with me and has my approval.

The act of 1885, Chapter 379 enables the Medical Society of the State of New York to change its by-laws and create membership upon such a

scheme as the Society shall approve, without application to the Legislature. Chapter 379 of the act of 1885 reads as follows: "Section I,—The Medical Society of the State of New York shall have the full power to elect such a number of permanent delegates or other members, as may be provided for by the constitution and by-laws of said medical society; said medical society being hereby empowered to regulate and control its own membership." "Section II,—All acts and parts of acts inconsistent with this act are hereby repealed." The Medical Society of the State of New York may amend its constitution and by-laws without further legislation by incorporating into its membership all members of the County societies. Therefore, the New York State Medical Association, if it shall so determine, may abandon its present organization and its members may become members of the Medical Society of the State of New York on a change being made by that Society in its conditions of membership. The transfer of the property of the Association may, under the authority of the Legislature being obtained, be transferred to the Medical Society of the State of New York. It would seem that if this consolidation shall take place, it is as important to the members of the Association as it is to the Society, that it shall be done without abandonment of the charter of the Medical Society of the State of New York, so as to preserve the continuity of the Society, which for almost one hundred years has existed under its original charter.

In all such societies, it is generally deemed one of the most important possessions to be able to trace back its history to the earliest possible time and all the members of the consolidated body will be equally interested in being connected with the early charter of the State Society.

If the Medical Society of the State of New York were compelled to seek a new charter, this Society would be obliterated and a new society would take its place. The only legislation necessary is that affecting the Association and its rights of property.

Therefore, in answer to the questions referred to me by Dr. Henry L. Elsner, Chairman of the Committee of Conference of the Medical Society of the State of New York, I answer as follows: Question I,—In order to bring about amalgamation of two bodies organized and chartered as are those which we seek to amalgamate, is it necessary for us to apply for a new charter?

To this question, I answer No.

Question II,—If we reorganize these societies under a new charter, do we cease to exist? In other words is there a moment of suspended animation?

I answer this question in the affirmative.

Question III,—If we need not apply for a new charter, must we still go before the Legislature to amend our constitution that amalgamation may take place, or has the State Society, under its present charter, power to take in the Association and change its method of organization to correspond with such changes as are finally agreed upon by both parties?

To this question, I answer, that it is not necessary to go before the Legislature to amend your charter. Legal union may follow whenever the Medical Society of the State of New York shall, by vote, change its conditions of membership.

Respectfully,

(Signed) CHARLES ANDREWS.

Dated, SYRACUSE, N. Y., December 17, 1902.

At the meeting of the American Medical Association held at Saratoga, June 1902, Dr. E. E. Harris of New York offered a code of medical ethics which was referred by that body to a committee of five. Section 2 of Art. IV of this proposed code reads as follows:

"The good of the patient being the sole object in view, any physician having a license to practice medicine conferred by a medical board authorized by the State may be aided in consultation."

Your committee would also call your attention to Section 3 of this same Act IV.

"No physician who indicates to the public that his practice is based on a sectarian system of medicine shall be entitled to professional fellowship or to recognition in medical bodies."

The constitution and by-laws of the American Medical Association adopted at an adjourned meeting of that Association held in Chicago immediately after the Saratoga meeting, no longer contains Chapter XV of the original by-laws. However your committee has the assurance of the President of the American Medical Association and the committee representing the New York State Medical Association that the old code of ethics of the American Medical Association is still in existence. This information was given to your committee at the meeting of the joint conference held in New York at the Academy of Medicine on Oct. 3, 1902, when the members of the Association once more asked your committee to subscribe to the by-laws of the American Medical Association.

Considering the radical action taken by the Medical Society of the State of New York over twenty years ago, your committee could not consistently take this step, nor could it suggest to your body submission to a code which changed conditions and the spirit of the times must of necessity efface.

At a meeting of the joint conference held in the city of New York on Dec. 19, 1902, the conference was honored by the presence of Dr. Frank Billings, the President of the American Medical Association, who journeyed from Chicago that he might be present and use his influence in favor of immediate amalgamation. Dr. Billings assured your committee that the old code was still in existence and unchanged. Dr. Billings also assured us, at the same time, that the Code of Ethics of the American Medical Association would not be considered binding upon State organizations.

Your committee, in spite of the argument in favor of immediate amalgamation, earnestly made by Dr. Billings and the assurance by him and the representatives of the State Association in affiliation with the American Medical Association that dropping Chapter XV from the By-laws of the American Medical Association did not eliminate the original code of ethics of the American Medical Association, could not consistently withdraw its objection and assured both the President of the American Medical Association and the committee representing the New York State Medical Association that the Medical Society of the State of New York was anxious to unite the profession upon a broad and liberal platform, one which it had reason to believe from the encouraging statements made by President Billings and the Association Committee the American Medical Association would adopt at its meeting to be held in New Orleans in May 1903, after which all matters of detail and recommendations to the respective bodies might be promptly and easily made.

In the unification of two powerful State bodies, like the Association and the Society, the method of organization becomes a perplexing question, requiring careful deliberation. Your committee feels that before final recommendations as to the method of organization of the reorganized Society can be made, it must come in the possession of further data. The will of the profession must be learned and given consideration. We ask for the power which shall bring us in contact with the individual members of County Societies that we may gather such information as shall, after mature consideration, lead to the formulation of a plan of reorganization. Among the plans of organization thus far suggested, we might mention: First,—The scheme by which all members of County Societies become members of the State body, thus abolishing entirely the delegate system. This reorganized State Society to have its various district branches, and a governing body to which shall be referred all matters of business including all ministerial functions.

Second,—Reorganization of the State Society which shall extend to the existing members of the New York State Medical Association permanent

membership with a continuance of our County Societies and the delegate system.

Third,—A plan which shall grant to the individual members of County Societies the privilege of permanent membership in the State body, not making such permanent membership in the State body obligatory.

Fourth,—A proposition will in all probability be presented to the American Medical Association at its coming meeting, by which all members of County Societies shall, for a single fee paid to the treasurer of the respective County Society, become members at the same time of the State and National bodies.

The proposed scheme toward which the American Medical Association has been lending its influence tends toward unification of the profession of the entire country and has for its object the extension of its influence and the enrolling in that body, through the county societies of all regularly educated physicians of this country.

These are a few of the suggestions concerning reorganization which have occurred to your committee. The question requires further thought and we believe that much valuable information can be gained by a thorough canvass of the State.

At the meeting held in the city of New York on December 19th, 1902, your committee with all its force objected to the resolution which was presented by Dr. E. D. Ferguson of Troy and unanimously adopted by the New York State Medical Association at its recent annual meeting. It reads as follows:

Resolved, That the report of the committee appointed to confer with a committee representing the Medical Society of the State of New York, for the purpose of devising a plan for the union of the New York State Medical Association and the Medical Society of the State of New York, is hereby approved.

Resolved, That the plan presented at the joint sessions of the two committees by the committee representing the Association, whereby 'the New York State Medical Association and the Medical Society of the State of New York be reconstituted by an act of the Legislature into a State medical body to be known as the Medical Society of the State of New York, of which all members in good standing in both bodies shall be charter members, and the reconstituted State medical body shall be the representative in this State of the American Medical Association by virtue of its acceptance of the Constitution and By-Laws of the American Medical Association" is hereby accepted by the New York State Medical Association as an expression of our sincere desire for a union of the medical profession in this State.

Resolved, That the committee is hereby continued for the purpose of cooperating with any committee from the Medical Society of the State of New York to secure a charter from the Legislature at its next session in 1903, which charter shall reconstitute the two State organizations into one State body, as set forth in the preceding resolution, but if the Medical Society of the State of New York shall fail to approve of such plan of union by a charter, to be secured at the approaching session of the Legislature in 1903, then this committee shall be considered as discharged, and the proposition of the Association withdrawn.

Resolved, In case this committee should find occasion to apply to the Legislature at its next session for the purpose of securing the said charter, it shall co-operate with the standing Committee on Legislation of this Association.

Your committee should not consent to the time limit insisted upon; it objected to the spirit of the resolution and believed that it was not in keeping with the earnest desire for harmony and unification so forcibly expressed at the various conferences with the committee representing the Association. Your committee was assured by the Association Committee that its individual members would be ready to use their influence in favor

of further conference after favorable action by the American Medical Association at its meeting in New Orleans.

The New York State Medical Association has during the past two years published a journal known as the New York State Journal of Medicine. During this time it has also published a Medical Directory of New York, New Jersey and Connecticut. Both of these have entailed an enormous expenditure of time and money. The Medical Directory is a worthy publication and speaks volumes for the energy of those who have placed it before the profession of these states. These publications have been losing ventures financially. Thus it will be seen, by reference to the report of the Treasurer of the New York State Medical Association, page 337 of the New York State Journal of Medicine, December 1902, that the total expense of publishing the Journal is as follows:

Total expense of publishing Journal	$3,051 73
Incidental expenses	93 29
Total	$3,145 02
Total receipts	2,168 20
Total cost to the Association	$976 82
The Directory account is as follows:	
Expense of publishing Directory	$4,967 54
Incidental expenses	651 97
Total	$5,619 51
Total receipts only	1,783 98
Cost to the Association to date	$3,835 53

It will be seen therefore, that the loss to the Association from the publications of the Journal and Directory is $4,812.35.

The expense of the business office of the Association during year 1901 was $5,693.18.

This included

Rent	$ 150 00
Salaries	3,799 84
Insurance	20 00
Office incidentals	1,723 34
	$5,693 18

The committee feels that if reorganized, the Society would not be able to bear these enormous expenses and could not therefore publish either the Directory or the Journal.

While we believe that an official directory of the medical profession of this State should be published annually, we are of the opinion that the work with its associated responsibilities might well be relegated to one of the many publishing houses ready and eager to undertake it, with facilities to make such a publication profitable.

The total expenditure of our State Society, including the publication of our Transactions, an unbroken file of which to-day represents a possession of which any physician may well be proud, is in the neighborhood of $3,000.00.

The expense of our business office, including the modest salaries of our Treasurer and Secretary are between $500 and $600.

Our committees expend about	$ 500
The Transactions cost, including postage	1,200
Cost of the annual meeting, between $400 and	500
Stationery, printing receipts, etc., between 200 and	300

The income of the Medical Society of the State of New York and the New York State Medical Association in the event of reorganization, would be insufficient to meet such expenses as would be necessitated by the publication of the Directory and a monthly Journal. It must be remembered that the receipts of the enlarged Society would not equal the combined incomes of the present Association and Society, for there is a surprisingly large proportion of fellows affiliated with and paying dues in both bodies, who in the event of amalgamation, would pay but a single fee.

In reporting progress your committee wishes:

First.—To file its objection to any method of reorganization which shall in any way interfere with the life of the Medical Society of the State of New York. Therefore we are opposed to any legislative act in connection with the amalgamation of the Medical Profession of the State of New York. The opinion of Judge Andrews makes it clear that no such action is necessary.

Second.—Your committee having been assured that the old code of ethics of the American Medical Association is still in existence does not feel that it is within its power to recommend that action be taken by the Medical Society of the State of New York, until the American Medical Association shall make it possible for the Medical Society of the State of New York to subscribe to its constitution and by-laws and written rules of order, consistently, without the sacrifice of principle.

Third.—The methods of gathering data which shall serve to formulate a plan of organization ultimately to be presented to this body for approval must be evolved after further conference and greater consideration than your committee has as yet been able to give to this important subject.

Fourth.—The Medical Society of the State of New York cannot continue to issue the Directory and the monthly Journal, now being published by the New York State Medical Association, in the event of reorganization.

A consideration of this report must prove to your body that this committee is in the midst of its work; that the many perplexing questions connected with the unification cannot be decided at this meeting; that there are many matters of detail which still demand attention and further conference.

For these reasons we beg leave to report progress; ask that our report be received and that further time be given to your Committee of Conference.

Respectfully submitted,

(Signed) HENRY L. ELSNER, *Chairman.*
A. JACOBI,
A. VAN DER VEER,
GEORGE RYERSON FOWLER,
FRANK VAN FLEET.

ANNIVERSARY ADDRESS

PROGRESS, LIBERTY, UNITY

By HENRY REED HOPKINS, M. D.
BUFFALO

THE submission of a free people to the executive authority of government is no more than a compliance with laws which they themselves have enacted.

The first requisite of liberty, as we and our forefathers have known it, is the willingness to abide by the law.

Fear nothing but to do wrong, dread nothing but to be recreant to public duty.

A sympathetic study of the history of our society, a history which began nearly a hundred years ago; an intimate personal knowledge of its affairs for almost half of that time; a review of the general progress of medical discovery during the same period; a careful study of its medical legislation and litigations; some acquaintance with the coincident developments in political, economical, and sociological science, and meditation upon the contemporaneous movements of certain great medical bodies toward organization or reorganization on broad, catholic, legal principles, have convinced me that the medical mind is prepared as it never before has been for a judicious consideration of the important truths included in our title.

The facts I ask you to consider fall naturally into three groups,—the political, the practical, and the scientific.

POLITICAL.—I know of no duty, next to the worship and obedience which we owe to our Father who art in Heaven, that is more imperative in its requisitions and more delightful in the performance, than that which the municipal law requires from its various professors. To be shielded and led by the divine nature and immortal reason of law is one of the most sacred rights of man.

It seems to have been assumed by the state from the beginning, and the assumption has been consistently sustained by the

courts, that as the state recognizes religion, but no particular sect, so it recognizes medicine, but no special school of practice. The state does not claim to be an expert in the fine distinctions of either religion or medicine, but freely concedes to the masters in both that priceless gift, the most precious in the power of any government to bestow, liberty—liberty in worship and in science. Our constitution provides that the worship of Almighty God shall be free in thought, in heart, and in form; that in medicine belief and practice shall be free; its investigation unhampered, its conclusion uncontrolled by human authority.

Only by grasping the full significance of these two facts: (1) that the medical profession is a unit; (2) that it is free and its members independent, and holding them in equipoise without slighting or minimizing either, may we prepare our minds for the right reception of the truths of this question, in sound discretion and for the furtherance of justice. So, and only so, can we prove that we have made good use of our liberty; so, and only so, can we demonstrate our fitness for American citizenship.

Our state constitution reads: The enacting clause of all bills shall be: The people of the State of New York, represented in Senate and Assembly, do enact as follows. We will do well to remember that the sanction and authority of law have lost nothing in force or in continuing effect by the transfer from the former style of We, by the Grace of God, King, Emperor, Most High Sovereign, Ruler and Potentate, to We, the people of the State of New York, grateful to Almighty God for our freedom, in order to secure its blessings do establish this constitution.

In the olden time obedience to law and reverence for it sprang from the conviction that the King's authority, the source of law, could do no wrong.

Shall not we who have enjoyed for more than a century the blessings of liberty repeat with equal fervor, with increased loyalty, with added appreciation, the words of Webster: Justice, sir, is the great interest of man on earth. It is the ligament which holds civilized beings and civilized nations together. Wherever her temple stands, and so long as it is duly honored, there is a foundation for social security, general happiness and the improvement and progress of our race; and the still more expressive apostrophe of Everett: Universal law—in action alternately executive, legislative, and judicial; in form successively constitution, statute, and decree—mingles into one harmonious, protecting, strengthening, vitalizing sublime system; brightest image on earth of that ineffable Sovereign Energy, which, with mingled power, wisdom and love upholds and governs the universe. Again, it is only necessary to recall that the

sovereign will of We, the people of the Empire State, has enacted that the medical profession shall have unity and liberty, and as we value liberty—that priceless blessing—let us recall that liberty is our birthright under the constitution, which we may magnify, honor and defend by obedience; and that effectiveness in progress, security in liberty, demand unity.

And further, we must not forget that responsibility is always measured by opportunity, that the strength and perpetuity of constitutional government are established and promoted in proportion as the whole people show sympathetic obedience unto the statutes and unto the judgments. We of the medical profession know more than the average citizen; knowledge increases responsibility. How may we better teach the ignorant artisan —half savage and part anarchist—self-control, moderation, respect and obedience to law, than by our own example of obedience, and of obedience even against inclination.

Moreover, I am sure that now, as in times past, misunderstanding plays a large part in the opposition of our profession to the efforts of the state in the direction of medical unity. We have feared that the state desired and sought to produce by legal enactment uniformity of medical practice. That is an impossibility. Men are individuals and individuals will differ. A clear perception that medical unity is not uniformity of practice, and that a proclamation of uniformity would be a denial of freedom and therefore impossible, should prevent further misunderstanding in this direction and enable us to appreciate rightly, legal medical unity, its origin, nature, and potentiality, to the end that we may conduct in harmony with its principles.

Unity in medicine is absolutely essential to efficiency and progress in that most important branch of sanitary science, state medicine. Consider for a moment some of the characteristics of state medicine. It is the supreme function of our profession. Success here means honor and confidence both at home and abroad and wins the sympathetic ear of the most liberal progressive and competent men. Nor should we forget that its work is never ended. Its problems increase in difficulty and complexity with the age of the government, the density of population, and the wealth of the people.

Sanitary codes,—the most intelligent and the most humane of legal enactments,—are the joint product of the will of the people and the wisdom of their physicians. Such codes and the manner of their execution are the most accurate exponents of the real civilization of their times; their conception, enactment, and execution demand good understanding and sympathy between the parties.

Sanitary organization should keep pace with the progress of preventive medicine, and so far as the conditions of the people permit, it should be the most complete, the most intelligent and the ablest of all in this age of organizations.

What could be more appropriate than that the Empire State by a united medical profession, in cordial sympathy with the law and with the source of law, should show the way to a sanitary organization commensurate in dignity and completeness of detail with her exalted position and noble purposes, and evolve sanitary codes so perfect that the humblest child in the state might receive the fullest benefit of the wisest, most modern, and most progressive, preventive medicine?

I should be false to this occasion and its high responsibility if I failed to remind you that the sanitary organizations of our state, as well as those of her sister commonwealths, are distinctly inferior to those of contemporaneous Europe in quality of membership, in official rank, in public estimation and consequently in effectiveness.

The wife of the humblest moujik is attended at childbirth by a midwife, whose license runs in the name of We, Nicolos Alexandrovitch, Emperor and Autocrat of all the Russias, Tsar of Moscow, Lord of Pskow, Grand Duke of Smolensk. The state board of health of Germany is under the presidency of the Chancellor of the German Empire. Is it strange that the great cities of that empire have less than one-third of the typhoid fever that annually ravages the cities of this state?

The literature of state medicine contains no history more instructive or more inspiring than the account of the sanitary measures by which Hebrew slaves escaping from the brickyards of Egypt were guarded during the whole of that marvelous march in the wilderness of Zin,—march not of a week, the time that seems to be the limit of strength and resources for our regiments,—but of forty years, the time required for the wisest sanitation to transform a mob of slaves into a nation of warriors, statesmen, architects, historians, poets, capable of establishing the civilization of Palestine, of building the temple of Solomon, and of producing a monument more enduring than marble or brass, the literature of Isaiah and of David. But the head of their board of health was Moses, the man who talked with God face to face.

Our minds should be stimulated, our convictions strengthened, our energies aroused by the contemplation of the fact, that the glorious accomplishments of state medicine in the past have been achieved without aid from the science of hygiene; our fathers had only faith and hope,—we have also demonstrated

knowledge. The introduction of pure water to the cities of Europe was not an experiment; the sanitary campaign of Wood in Cuba was not an experiment; state medicine of to-day is not an experiment, and investments under its direction are not speculations.

HEAR THE CONSTITUTION.—We, the people of the United States, in order to form a more perfect union, establish justice, insure domestic tranquillity, provide for the common defense, promote the general welfare, and secure the blessings of liberty to ourselves and our posterity, do ordain and establish this constitution for the United States of America—to promote the general welfare.

The efficient sanitary organization of our country, where the humblest citizen is a member of the royal family, will not be suitably inaugurated until there shall be in the cabinet at Washington a secretary of health. The medical profession of the United States would require but a few national campaigns in the name and interest of State Medicine to accomplish this most salutary, most desirable of sanitary achievements.

PRACTICAL OBSERVATIONS.—The absolute justice of the state enlightened by the perfect reason of the state—that is law.

Reason is the life of law; nay, the common law itself is nothing else but reason.

Let us consider the possibility of having in our state a medical profession, united and organized for the purpose of State Medicine, and for such other purposes as experience and wisdom may deem advisable.

What are the essentials of such an organization? They are three: (1) a common belief in principles so clear, so cogent, so logical, so practical that it will engage our minds, strengthen our convictions, stimulate our ambitions and warm our hearts until conviction may ripen into action; (2) an occasion calling for the efforts of such an organization; (3) men to engage in the work who are urged on by a conviction of its necessity and eager for the labor it demands.

The art and science of State Medicine, the present needs of our people, our enormous death-rate from diseases known to be communicable and therefore preventable, the necessity of a higher standard for state license, furnish the occasion for the required opinions, knowledge, and convictions.

We are here. We are officials of the government. In the mind of the state we are one. We are working at the problems of state medicine, and in spite of our lack of unity we have made some progress toward their solution. We still believe that in union there is strength. The proposed unity, without requiring

us to surrender any principle, prerogative or opinion, furnishes an opportunity of showing to the world a conspicuous instance of obedience to the law, of deference to the will of the sovereign people at the cost of some small sacrifice of custom, prejudice or pride.

It is significant that medical unity is tacitly recognized by ourselves as present in a greater degree than some would admit —from filing a certificate as to the cause of death all the way up to the highest function performed by a medical man, service upon a board of state examiners for state license.

We may well give a moment's consideration to the frame of mind on our part which is essential to a profitable consideration of this subject. Two things seem to be needful: (1) a thorough understanding of the temper of our legislators expressed in the medical laws of the state, together with an intimate knowledge of the law itself, and of the philosophy which underlies it and will be sure to influence any and all of its future developments and amendments. There is not time to treat the subject exhaustively and I can only advert to a single principle which seems to me the more important. The theory of our medical law has been, is, and will be, that to the people the medical profession is a unit. There may be, as there have been, varieties of belief, but there must be a common responsibility. The law never has undertaken and never will undertake to discriminate and choose between different medical beliefs and opinions regarding the mode of action of a given drug, or the method of treating a given disease. Upon such questions the law is silent, justly discerning that they are for the decision of medical men, singly, or in assemblies where varying minds and varying experience may hear and determine, and where if wrong conclusions are adopted, further observation, further experiment and fuller light, may make it the plain duty of to-day to reverse, as so many times has been done, the findings of yesterday.

Again, it seems to be essential that we should show a sympathetic loyal obedience to the law of the state both in its letter and its spirit. These words sympathetic, loyal obedience, are not lightly selected, but seriously chosen, for the obedience in question must be from the heart; it must be a sympathetic obedience, not the obedience of the officer who hates the regulations he is forced to observe, but that of the loyal Jesuit who obeys because he loves.

Finally, to this knowledge of the law, and to this condition in our hearts of sympathetic obedience thereto, must be added as a requisite to that state of medical organization, whereby we alone may meet and discharge our full responsibility as the

guardians of the public health, an abiding conviction of the need of medical unity. Remember that in stewards it is required that a man be found faithful.

Only to those of us fortunately possessed of these qualifications of knowledge, mind and heart, is this argument addressed; only to such does it seem possible that a wise conclusion may follow a judicious consideration. To others it will be like attempting to present the arguments of Christianity to those who disbelieve in the existence and immortality of the soul.

Again, it may be stated as a general principle, that medical unity will never be made easier, brought nearer or made more efficient, by dwelling upon the limitations, or the imperfections in conduct or practice of our brethren. As long as we remain men such limitations and imperfections will abound. We ourselves may possibly at times show this evidence of a common humanity, and our brother's infirmity is but one more reason why he should have the advantage of our strengthening and elevating society.

Wisdom and experience teach that it is expedient for us to make much of our brother's good qualities, and that as to his faults forbear to judge, for we are sinners all.

We have seen that a successful organization must have the continuous stimulus of work which calls for the doing, work which must be done, work which may be done only by the organization in question.

Does such exist here and now? Surely it does. First of all, the best efforts of the organized medical profession of this state are needed to extend to its millions of people the protection, the preventive protection, of the demonstrated knowledge of hygiene.

It may not be our proud privilege to be pioneers and discoverers of new truths in any of the many departments of hygiene, but at least it is our duty to keep abreast of the column of progress and discovery, to be ready to receive the advances, to take advantage of the discoveries, to transmit to our people the priceless sickness-preventing and life-saving hygienic truths which are being coined from day to day by the army of scientific pioneers,—remembering that we are just entering upon a new century, and that the highest round of successful accomplishment in one age is frequently only the point of departure for that which is to come.

The limits of your courteous forbearance and consideration for the proprieties of the occasion, do not permit an extended presentation of the manifold questions of hygiene that now press for solution. An entire address might well be devoted to the consideration of a single topic. It is enough to say that each

is important, and that the whole constitutes a body of work, of transcendent moment, work in a realm in which the coin is not silver or gold, but that infinitely more precious material—human life.

Let me invite your attention to a group of communicable diseases which differ in destructive power and in the manner of communication, but are alike in their diffusion and alike in the manner in which their prevention is neglected,—gonorrhea, syphilis and consumption. From this one group, mentioned in a single breath, we suffer an annual mortality and stagger under an everlasting load of affliction and maiming almost incredible.

To attack this single group of diseases with success, demands knowledge, resources of legislation and command of capital in sovereign amounts. Here is an opportunity for every physician and philanthropist in the state to engage in work which can be accomplished only by and through the most thorough of organizations.

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Intimately related to the foregoing in more than one way is the interesting and important sanitary problem of a better housing of the poor in our larger cities.

This subject is already a nightmare to those who study the causes operating to form the congested areas in our growing cities, and who realize what a menace to health and morals, to right living and to good citizenship such congested areas always are. Here, as in the previous problem, resources of the most varied character, ability of the highest order, zeal and perseverance second to none are required. Three thoughts should move us to prompt action: (1) the belief on the part of all students that prevention is easier and more economical than cure; (2) the conviction of the best informed that in many instances the growth of our cities has but begun; (3) the general consensus of feeling that the present century is to be noted for its success in the attack of hitherto resistant sociological problems, and that of such problems the better housing of the poor in cities is of preëminent importance.

For years those having authority have directed attention to the impure state of the waters in many of our streams, and the consequent prevalence of typhoid fever not alone, or chiefly, in large cities, but in many of the smaller towns. Attention has also been called over and over again to the important relation between the health of the community as well as to the productivity of the soil, and the protection from floods of a due proportion of forest lands. We know that in nature's great storehouse, where provision is made for the preservation of health and

restoration from disease, no items equal in value pure water, pure air and sunshine. To keep our streams and other sources of water supply free from foulness and infection, to preserve our forests from useless destruction, to redeem denuded regions by reforestration, to drain our marshes where breeds the disease-bearing anopheles, to transform the foul disease-spreading dampness of the sub-soil in our city streets into the benign, protective, ornamental foliage of the Carolina poplar, the elm or maple,—this is a work worthy of kings, a work that only kings may do, a work that the sovereign people of the Empire State may only accomplish when inspired and led by the united profession of medicine.

Without doubt, the most important event in our medical history of the last half century was the institution of state examination for license to practice the healing art. The immense significance of state license is in the fact that there we see the coöperation of the sovereign people and the united medical profession, the laying on of hands of pontif and profession, the blessing and the commission of the majesty of the law, the will of a free people, blended and united with the spirit of the art of healing, the most humane among all the arts and sciences; the recipient of the state license bears an honorable commission to a noble vocation.

Agreeable to the legitimate expectation, coincident with the establishment of state license to practice medicine an evolutionary revolution took place in the medical schools of the country. Before the time under consideration many of the graduates of our medical schools were prepared to enter upon an honorable career in medicine, but some of the graduates of such schools were indifferently prepared to get an easy living in what they supposed to be a lucrative trade. To prepare men for the medical degree of the college was one thing, to prepare them for the state examination quite a different one. For the former, two short terms and a few—less than a dozen—teachers would suffice; for the latter, four long terms and many dozens of teachers were required.

For the former, a medical faculty in every hamlet was the cry; for the latter, let us unite and found a great university is the word, and the joining of hands of the sovereign people and the united profession in state licenses is but a thing of yesterday; of a single decade. We may be well assured that no single responsibility is comparable with that of cherishing and developing and extending the principle of state examination and license; to it must continue to flow our best thoughts, our highest endeavors, for from it must come, if come it may, that high

standard of personal and professional excellence and preparedness which alone may enable us to cope with the ever increasing intricate problems of modern medicine.

This commanding occasion gives me an opportunity to remind you, that there are in this state three examining boards where there should be but one; that the responsibility for this useless multiplication of official machinery rests primarily upon this society; that the people are aware of no good reason for this confusion; have but a scant and grudging patience with it, and that it is within our power, and in my judgment is our bounden duty to take the proper steps to make medical unity in state examination, unity in form as well as unity in spirit.

With a single board of examiners, representing the entire medical constituency of the state,—the entire lawful profession of the state, and therefore supported by the united medical profession,—we would be for the first time in position to expect and require of state examination a higher and still higher performance of its discriminating and selective functions. To some of the further practical demands upon our system of state license, demands of to-day, let us now for a few moments direct our attention.

I am informed that it has been from the first the wise policy of our state boards to raise the standard of examination from year to year slowly, but steadily; this course has without doubt been conservative and wise, and the large proportion of rejections indicate that the work of our teaching bodies is still far below the standard of the licensing body. However, it may be observed that the present standard of examination for license is not greatly different from that which obtained when the examination was inaugurated, whereas the provisions of our teaching faculties between then and now have changed greatly. Then terms of three months in two years, with lectures from a few teachers was the rule; now terms of six months, for four years, with instruction from scores and scores of teachers is the mode.

It is not apparent, that the time has come, or will soon come, when it will be expedient to raise the standard for license in this Empire State so high that we may with truth declare of it, that there is none higher?

At present the severity of the test exacted by our examining boards of candidates for the state license is distinctly less than that for entrance to the medical service of our army, navy or marine hospital corps—is distinctly less than that of state examination or licensing examination in the chief countries of Europe. And yet the subjects of Edward or of William, of Italy or of France, neither deserve nor are able to reward better medical

services than do the citizens of this state; it would be an easy and delightful task for a single state examining board supported by the united medical profession to raise the efficiency of that profession in this state so high that all might know and feel, that in illness, in accident, or in childbirth, every citizen, even the humblest dweller in the city tenement or in far away forest log house, would receive the care and attention of a medical man of unquestionable ability.

Again, it is to be noted that the medical service of our state is by no means small in number or insignificant in importance. That a special examination should be held to test the fitness of candidates for this service, is a question deserving serious consideration. Possibly the growth and multiplication of specialists has not yet reached its limit and the specialist in hygiene may yet appear.

Certainly it is an important part of the duty of state examination to provide for the highest possible efficiency in all departments of the medical service of the state. The need of this requires no further argument than the bare enumeration of the more familiar of these positions. Our health officers, state and local, are charged with enormous responsibility, in the direction of patronage, the disbursement of funds, the protection of health, the prevention of disease; they are not known to be especially fitted for their onerous duties. It is generally believed that their efficiency might be increased. Our commonwealth and municipalities annually expend enormous sums in the support of institutions for the treatment or maintenance of the blind, the deaf and the dumb, the orphan and destitute, the insane, and the criminal; the medical service of these institutions is of vast importance pecuniarily, professionally, sociologically, and these medical men and women have received no special preparation for their work. Who will say that such special preparation might not well be undertaken, and that special fitness might not be properly tested by special examination by the state examiners?

Again, it is a matter of common knowledge, that the work of our coroners, and post-mortem examinations made for our coroners, do not always show that order of expert preparation desirable. Undoubtedly economy in the administration of the law, precision in the determination of medico-legal cases would result if the office of coroner was always filled by a medical man especially prepared for his work; and medical men at least would have more confidence in the reports of autopsies if they knew that men authorized for such work had made special preparation, and had given evidence of fitness, by passing with success a special examination by the board of state examiners.

Finally, we may not conclude these observations without adverting to the growing interest on the part of people and profession in the subject of school hygiene, and of medical inspection of our schools to the end that the health of the scholars and teachers may not be prejudiced by unhygienic conditions, and that the schools may not become the centers of distribution of the more common communicable diseases by reason of the attendance of pupils having the same. Here is work of singular importance, that possibly only awaits the appearance of the men and women properly prepared for it, whose fitness has been ascertained and certified by the proper authority.

It is confidently believed that the several subdivisions of medical practice before recited, constitute what might easily grow into a specialty of commanding importance, and of particular interest to the state and to preventive medicine, possibly sufficient to warrant the notice of our teachers of hygiene and of our state examiners. These practical observations are submitted for your consideration in the profound conviction, that the time is ripe for a sanitary organization to engage in the work of forwarding the interests of the public health, to include the joined forces of the sovereign people, and the united medical profession of the Empire State.

SCIENTIFIC.—The diagnostics and the prognostics of the maladies of nations, these are the largest questions pertaining to this world, that can be pondered by the mind of man.

That all the dead, the living, the unborn, are one moral person, one for suffering, one for action, one for responsibility.

There is that in American character which has never yet been found unequal to its purpose. There is that in American enterprise which shrinks not, and faints not, and fails not in its labors.

Your attention has been invited to observations of a more or less political nature, and again to those of a complexion more distinctly practical; it remains to lay before you certain conclusions arising from a contemplation of our subject upon a background of the science of medicine.

Let us consider the remarkable development which has recently taken place in our medical schools, a development which has already worked a substantial transformation in our teaching faculties, their equipment for work, and their mode of teaching. Without question this development is normal, is growth, is the product of natural forces, is to be permanent, and has but fairly gotten under way. In connection with this, three thoughts deserve our earnest attention.

Medicine, as practised at the bedside, or in the consulting

room, has ever been a blending of science and art; the mode of teaching medicine from the days of Hippocrates to the last decade of the last century has been that of men who saw and hoped great things of the art and knew but little of the science. Our profession, nevertheless, has been free in spirit and has profited immensely by the contemporaneous advances in the allied sciences of chemistry, physics and biology, has contributed to the glorious galaxy of heavenly bodies a star of the first magnitude—the science of bacteriology, and by the light of this new luminary is rapidly advancing in the hitherto most discouraging of all her fields, and is day by day adding triumph to victory, triumph in diagnosis, and victory in treatment. With the slow but surely increasing illumination of this department of practice, by the rising sun of scientific therapia, medicine may declare herself to be scientific in all her ways. The long and honorable era of the empiric teaching of the art of medicine has closed; the twentieth century finds the era of the demonstrative teaching of the science of medicine auspiciously inaugurated.

The importance of medical unity and of relations of confidence and sympathy with the people becomes manifest when we contemplate the cost for equipment and for maintenance, of numerous teachers, the vast libraries, the museums of specimens anatomical, physiological and pathological, the laboratories, chemical, biological, pathological and bacteriological, together with the facilities of hospitals and dispensaries, some of the prime necessities for the teaching of medicine of to-day. From Hippocrates to Koch is a long stretch in time and in method, but it is not greater than the difference in method and equipment between the medical college of the nineteenth century and the medical department of the university of the twentieth century.

Heretofore, we have directed your attention, not without conscious pride in the opportunity, to the fact that the spirit of medicine is free, that she wisely borrows, to profit withal from the discoveries and advances of the contemporaneous sciences. During the final decades of the last century the science of psychology has come into prominence, and has made invaluable contributions to the ancient and honorable art of pedagogics.

In part as a result of this, and in part from independent observations, the theories and the modes of teaching of to-day differ widely from those of yesterday—methods have changed greatly and will yet change. My knowledge of the methods of teaching current in our various medical schools is not sufficient to enable me to inform you how far medical teaching has bene-

fited by the current advance in pedagogics, but it is my great privilege to be permitted to congratulate you upon our fortunate situation. The University of the State of New York, the inspiration and the head of our magnificent educational system, licenses such of the graduates of our medical schools as successfully pass the state examination. As directed by law, accurate record is kept of the standing of those examined, with records of those who fail to reach the standard requirement.

These records from year to year throw a beam of white light upon the methods, efficiency and success of our different teaching bodies, emphasizing those whose pedagogical methods are deserving and winning success, and giving admonition and warning to others; that the struggle for existence is not confined to plants and animals, that in medical colleges only the fittest are likely to survive, that if our state standards of this decade are sometimes found to be inconvenient, those of the next decade may prove to be prohibitive. The question of medical teaching which shall be in accordance with the best medical traditions and principles, and also along the best pedagogical lines, is closely related to the question of large foundations, of endowments of chairs and scholarships, and this in turn is vitally dependent upon the sympathy and good-will of those who are rich in this world's goods.

Hitherto, mention has been made of the tendency of the times to develop for purposes economical, commercial or financial organizations and combinations, involving capital, employment, and product in quantities and proportions well-nigh fabulous, certainly beyond the wildest dreams of the men of business of half a century ago. This condition is not accidental, neither is it as some of the more timid assert, a sign of degeneracy of the body politic; but it is the legitimate result of free institutions under auspicious environment operating to produce and to accumulate the kindly fruits of the earth for the betterment and the enjoyment of the people, in such surprising profusion, in such enormous quantities as to verily indicate that this is the very promised land—that when our forefathers and our brothers shed their blood that there might be liberty and union on this continent, they did in fact and in confident faith seek the Kingdom, believing in the promise—and all these things shall be added unto you.

Of this we may be assured that as history will describe the last century as the age of preparation, of accumulation, in like manner the present century will be known as the age of distribution, of performance, of accomplishment. Indications are not wanting that the American stage is being set for the perform-

ance of an honorable part in the drama of life. Certainly wealth is accumulating here as nowhere else in the world, and emigration is pouring into our country a vitalizing, energizing, progress-making stream of human life, all with upraised faces, of greater significance, of more hopeful potentiality than the combined products of our mines, our mills and our farms. Again it is to be observed, that the wealth of America is a consecrated wealth, consecrated in the highest and most intelligent degree to the beneficent purposes of constitutional government. The benefactions of our people in the last decade for charitable, educational, scientific and allied purposes, is an almost incredible sum, and is a plain indication that the enormous wealth of the country is in the hands of men of high and noble principles.

The problems of the new century are before us. Which of these problems compares in interest, in significance, in importance to the welfare of the people, the maintenance of free government, to the progress of our race, with the scientific problems of state medicine?

How may we better prepare to meet those problems, than by striving for medical unity; hoping thereby to gain somewhat in numbers and influence and to improve distinctly in form—that our purposes may be the better understood by the people, that our institutions may be prepared to train men to the full stature of the science and art of medicine, that our legislature may enact suitable laws for the due protection of the public health, and furthermore that the sympathy and interdependence between the profession and the people may become so intimate that our sanitary codes and ordinances shall be everywhere intelligently obeyed.

Let us hear the conclusion of the matter. *E pluribus unum! Excelsior!* The spirit of America. The thought of the Empire State. The Zeitgeist of the twentieth century. The science of medicine. Never before in the world have these mighty thoughts carried such possibilities of duty, of conviction, of action, as now. *E pluribus unum!* Our theme is the all sufficiency of the law, concerning which it has been written, Of law there can be no less acknowledgement than that her seat is the bosom of God, her voice the harmony of the world; all things in heaven and earth do her homage, the very least as feeling her care, and the greatest as not exempted from her power. *E pluribus unum!* Thus, for more than a century. We are many, we may be one. We are many, with all the indirectness, the confusion, the inefficiency, that ever attend disunion and disorganization. Let us become one, one for the defense of the public health, one for the uplifting of every member of our profession, one for

the promotion of the most humane, the most inspiring, the most beneficent of all material vocations, the science and art of medicine. *Excelsior!* When a regiment advances a company must take the lead. You hold in your hand a glass containing a solution of a given salt of a certain concentration. Your desire is that the salt shall evolve from the chaos of solution to the accurately, efficiently, organized form of crystalization. You drop into the glass a single crystal of the given chemical and in due time chaos gives way to order, solution yields to crystalization, disunion and disorder give place to union. The medical profession of America is without unity, or such organization as becomes the free profession of a free and united people. What could be more appropriate, more inspiring, more eminently distinguishing, than the example of the Empire State, the state of the metropolis of all the Americas, the state of Alexander Hamilton, of Chancellor Kent, of John Jay, the state of enormous wealth, the state of marvelous achievements, of vast and bewildering possibilities, giving to the profession of America, an example of medical unity under the law!

The science of medicine! Who of all our number does not feel his heart quicken with pride at the very sound of the words? Who of us in years past middle life does not recall the heart-sickening horrors of our early practice, when confronted with the signs of early pulmonary tuberculosis, realizing its inevitably fatal termination; or of laryngeal diphtheria in a child under five years of age, knowing our helplessness, and the hopelessness of the case, and the agonizing scenes of the slow suffocation to death from laryngeal stenosis; or the signs attending acute peritonitis, remembering from sad experiences, from reading, and from instruction, the enormous proportion of fatalities in the wellnigh hopeless malady? How thankful we are that having lived to see and endure so much of the uncertainty, the helplessness, the hopelessness of medicine of thirty years ago, it has been graciously vouchsafed to us also to live and to work in a period when science has put into our hands such vast powers of making early and accurate diagnoses, and modes of treatment, surgical and therapeutical, of such immense power and life-saving efficiency. Thanks! and thanksgiving to Almighty God, just and honorable pride in our noble profession, laud and praise and everlasting honor to the men who have made, who are making, and who are yet to make the science of medicine; these sentiments are becoming and appropriate to the subject and the occasion.

ADDRESS

RUDOLF VIRCHOW; AN APPRECIATION

By CHARLES A. L. REED, M. A., M. D.

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CINCINNATI, O.

THE object of this address, the invitation to deliver which is an honor for which I am profoundly grateful, is to express, in some measure, an appreciation of the life and labor of Rudolf Ludwig Karl Virchow, a deceased Honorary Member of the Medical Society of the State of New York.

In approaching this task, we become at once impressed with the fact that the influences which develop greatness are subjects of speculative inquiry not less interesting and important than the momentous question of what constitutes greatness itself. When, therefore, we for any reason examine into the facts relating to the evolution of a given historic character, we at once think of the conditions and forces concerned in its production—we think of ancestry, of domestic surroundings, of scholastic opportunities, of personal associations, and of the forces that were at the time dominant in the social, political and intellectual atmosphere. We are prone, also, as we turn to greatness itself, to measure it, not alone by the standard of its own time, not alone by the rule of personal achievement, but to estimate it with reference to both its immediate importance and its final influences. Thus, as we glance over the vista of history, and our fancy nestles naturally enough about the most imposing figures of the ages, we discover, for instance, that we would like to know more of John and Mary Shakespeare, who blessed mankind with the Bard of Avon—the man of sympathy; and we yearn for an acquaintance with the farmer of Woolsthorp and his wife who begat the illuminating genius of Newton—the man of mind. We feel, also, that, full as are the annals, we would like to know even more of the actual political forces of the Roman Empire, directly concerned in developing the world's greatest soldier and statesman; that, notwithstanding its rich literature, we would like to feel the sentient throbs of the Elizabethan epoch that made possible the world's greatest poet and dramatist; that, voluminous as is the record, we would like to be more familiar with the trend of scientific thought during the century that followed and that produced the world's greatest philosopher. We search for the reason why each of these names has been engrossed

upon the scroll of immortality, and as we search we discover that everywhere lies the unknown; that, in atom and planet, in germ and genus alike is law, natural law, inherent and integral, whose essence is not and cannot be of the record; that these men have delved and found and revealed laws—laws of war and statesmanship, laws of human emotion, laws of the natural universe—and that man is stronger and better and happier because they lived. We discover, furthermore, as we glance over that immortal scroll, that each name inscribed thereon, has been placed there because its possessor, whether Pythagoras or Euclid, Copernicus or Kant, Galileo or Herschel, Hippocrates or Harvey, whether Dante or Goethe, each has been recorded because each has torn away the veil and revealed somewhat of the law that was hidden; for the law is in all and of all, and he is the greatest among men who reveals the most of law unto man. In this spirit let us approach a discrimination of the great savant whose demise was the melancholy event in the medical, the scientific and the political world during the last year.

The Second Peace of Paris had been signed but a few years when, in 1821, Virchow was born in the little hamlet of Schievelbein in the flatlands of Pomerania. The Baltic breezes that swept inland on that fifteenth day of October were not, however, sufficient entirely to cool the political atmosphere of that northernmost Prussian province, or, for that matter, of the thirty-nine petty states that then comprised the German Confederation. Napoleon's exile had terminated with his death at St. Helena but six months previously, and Europe—Germany in particular—relieved of the depressing shadow of his overlordship, was busying itself with the always serious problems of reconstruction. The intellectual world was not less perturbed than was that of politics. The great universities, then as now, exercised a powerful influence upon the trend of events. It was in them that the battle for constitutional rights, as a remedy against the despotism of the petty Princess, was waged with intensest vigor. A constitution had been granted and revoked Würtemburg; the Duke of Weimar had granted a constitution to his subjects, the celebration of which event, at Jena, had led to patriotic demonstrations and to a revival of the influences of Luther's great struggle for liberty of thought. The movement thus engendered had become so formidable as to invoke the oppressive antagonism of the tyrannous Metternich and the promulgation of the infamous Carlsbad Decrees, which provided for the rigorous censorship of the universities and newspapers by Government commission. Their provisions involved the suppression of any newspaper and the exile of any man who might

express opinions inimical to the policy of the Government. The same interference with free thought existed in Austria and in Lombardy. The students of the University of Turin had been massacred because they appeared at the theąter in red caps. France and Spain were in a state of unrest, and among the countries of Europe—the people—from the Mediterranean to the Baltic, the effort was being made to conform human life to those inherent laws of the social fabric that most make for happiness. The effort to reduce these laws, natural, inherent laws, to definite terms; the effort to adjust habits and customs to new ethical rules and to new constitutional provisions, produced a state of mental activity and of moral daring in every part of Continental Europe. This, then, was the social, political and intellectual atmosphere that prevailed in every German home, and even in the homes of Karl and Johanna Virchow, as they rocked the cradle of him, the formal appreciation of whose long and illustrious life is the object of our solemn reunion at this hour.

The clamor against absolutism was heard in childish murmurs at the public school at Schievelbein, to which young Virchow went at a tender age. There, in the little town in which the Reformation had long been the dominant force; there, in the little school beneath the shadow of the church, the synagogue, and the Castle of Malta,—a combination that in its catholicity was almost prophetic—the youth encountered forces that were potent in fashioning his subsequent illustrious character. It is in this fact that we, in free America, where the schoolhouse stands as the temple of rational belief, where it stands as the safeguard of the Republic, may take peculiar satisfaction.

The political agitations of the times, the little rivalries, the little hatreds, the fierce combats of the public schools, were not, however, sufficient to divert the youthful pupil from the successful prosecution of his studies; for, we learn, that he went, under age and with a particularly advanced knowledge of Latin, to the Gymnasium of Koslin, where he was a source of surprise to the Director. Here, at Koslin, again was the spirit of the Reformation, with its inspiration of truth and liberty, and its yearning for happiness. The fact was recognized even as far north as Pomerania that, in the Rhenish provinces, previously ruled by French officials, there was a higher idea of human rights than obtained in the other provinces of the Confederation, especially under those ruled by the powerful House of Brandenburg. There was, therefore, a clamorous appeal for the recognition of all that was attractive and great in the principles of the French Revolution, and the outcry for a constitution embodying those principles came from no province with more emphasis

than from Pomerania, and from nowhere in Pomerania with more insistence than from Koslin and from Schievelbein. The Revolution of 1830 had brought coveted charters of liberty to Brunswick, Hanover, Saxony and Hesse-Cassel, while to Prussia it had brought only the farcical concession of a system of triennial, provincial diets with merely consultative powers. In spite of these distracting influences, however, influences that are always alluring to the enthusiasm of youth, young Virchow passed from the Gymnasium in 1839, first on the list of the Abiturienten. The independence by which industrious and ambitious youth refuses to be restrained within the confines of an arbitrary curriculum, is always the prophecy of a broad manhood. The child, in this instance, and by this rule, was, indeed, father to the man, for we find that he presented himself for his finals, not only in the required branches, which were difficult enough, but in Hebrew, which he had mastered from pure love of philologic research. It was this same impulse that prompted him, during the succeeding few months, to master Italian without a teacher, just as years later we find him resting himself from his scientific labors by delving into the charm of modern Arabic poetry.

A few months after leaving the Gymnasium he set out for Berlin, a journey which, in those days, before the introduction of railroads, had about it more of adventure than is involved in the two hours' run of to-day. Of his career as a student in the Frederic Wilhelm's Institute, it is sufficient to say that he was an arduous student. In the faculty then were Dieffenbach, the foremost surgeon of the day; Schönlein, who had come from Zurich the same year to join, not only the teaching body, but to act as reporting council for the ministry and to serve as physician-in-ordinary to the King; Froriep, who was in charge of the Pathological Museum at the Charité, and who, in addition, served the Government as Medical Counselor; Caspar, who was also a Medical Counselor, with a seat in the medical deputation for medical affairs in the ministry; but, towering above all, was the intellectual figure of Johannes Müller, the professor of physiology. He was an original genius, with daring, actually engaged in winnowing the wheat of demonstrable truth from the then prevailing chaff of egoistic opinion—to divorce a physical science from speculative philosophy. Prompted by the inspiration which he had derived in turn from Bichat and the French school, this professor of physiology was busily engaged in retesting in the laboratory truths previously elaborated by Haller, Whytt, Spalanzani, Cullen, Prochaska, John Hunter, the Bells, Magendie, Berzelius and Bichat himself. My fancy

likes to dwell upon the almost dramatic moment when the shopkeeper's son from Schievelbein, the little keen-eyed, yellow haired stripling of nineteen, was ushered into the presence of this, the great founder of the modern school of physiology. There was in that meeting an intellectual impact that resulted in the transference and the perpetuation of great thoughts, great methods, which, perfected by the pupil, lead to still greater results. It was from this great professor that Virchow, during the next four years, was to derive those habits of investigation which, coupled with the spirit of daring, was to make him, in turn, the leading investigator in the realm of biological research. It must be remembered, however, that with all of the social and political disturbances, Germany was at that time thoroughly impregnated with a wholesome ferment. It consisted of the spirit of rational investigation, and was infused by Liebig in chemistry; by Humboldt, who was promulgating his discoveries leading to the publication, five years later, of his Cosmos; and by Froebel, who was establishing his marvelous principles of education derived from Pestalozzi, and which have since borne rich fruit the world over in every department of human instruction. It is not surprising, therefore, that, with these antecedent influences, with these present surroundings, with these dominating forces, and with his marvelous insight and industry, Virchow should make such a record as a student, that upon his graduation he should be given the assistantship to the Prosector of the Charité Hospital. It was his first recognition, and it came with deserved promptitude. He was actuated at this time, as in his entire subsequent career, by the broadest principles of catholicity. During his student career, in addition to the prescribed lectures, he had gone into logic and psychology; in his busy, energetic way, he had mingled with the political organization among the students, and there were already manifest tendencies which, a very few years later, brought him before the German public as a scientist, a philologist and a social reformer, and a democrat. Promotion came with undue delay. Froriep resigned as Prosector in 1846, and Virchow was elected to the succession. His work with Müller, however, had brought him in contact, not alone with that great man's scientific method, but with his habits of publicity, as a scientific writer. *Archiv f. Anatomie, Physiologie u. wissenschaft. Medicin,* long issued by Müller, soon found an imitator in the Department of Pathology in the periodical issued jointly by Virchow and Reinhardt, and which, on the demise of the latter, Virchow continued to edit until his death. The political and economic conditions were fashioning themselves into the Revolution of 1848, when Virchow, already in

influential touch with the Prussian Government, was delegated to investigate an epidemic of typhus fever which was then raging in Upper Silesia. The work was done with his characteristic thoroughness, transcending the prescribed limits of his instructions. He investigated not alone the pathological and clinical phases of the disease, but he entered freely into a discussion of the hygienic, economic and social conditions underlying the epidemic. He even went so far as to indicate a number of social reforms, essential to the prevention of such epidemic and tinctured his science with considerable democracy, his outspoken utterances, in these particulars, causing a distinct sensation in the Ministry.

The trip to Silesia seems to have been a very important experience in Virchow's career. His previous tendencies as a reformer in the direction of popular liberty, were now fully confirmed, and he became an active participant in the great revolutionary movement of that year. He unhesitatingly promulgated his platform as that of full and unrestricted Democracy, on which theme he made violent speeches to the Berlin populace, by whom he was elected a member of the National Assembly. His political ambitions, however, were destined to be temporarily curbed by the fact that he was under the parliamentary age, and was, consequently, not permitted to take his seat. His energy, however, found a compensatory outlet for, with Leubuscher, he founded a journal which they called *Die medicinische Reform*, through which he advocated the establishment of a Ministry of Health and insisted, among other measures, that medical education should be made free. These suggestions, not originating with the government, were scarcely less distasteful to the Ministry, than was his report from Silesia, or than were the political harangues which he continued to pronounce to the plaudits of his fellow burghers. He seemed at this time to be largely dominated by the spirit of iconoclasm, not, however, that form of iconoclasm which is merely an expression of the spirit of destruction, but that better iconoclasm by which old gods are destroyed that newer and better ones may be erected. He invaded the realm of theology, and proclaimed, not a mere agnosticism, but a positive disavowal of the existence of a Hell, insisting that "only a benighted Mechlenburg pastor could be so foolish as to believe in a Devil." The Government, committed not only to the task of maintaining the national order, the national laws, but the national religion, looked upon the young orator as a dangerous polemic. He was compelled to resign his appointment as Prosector for the Charité, where, in spite of all his political agitations, he had conducted epoch-making re-

searches on leukæmia, embolism, thrombosis, phlebitis and other phases of morbid anatomy. He had already become a teacher of ability, and his researches had attracted widespread attention. These facts, quite as much as the influence of his colleagues at the Charité, probably saved him from the decree of exile issued at that time against many participants in the revolutionary movement. He was, however, banished from Berlin to Würzburg, where, in May, 1849, he accepted a chair in the faculty of the University. There was here less opportunity for effective participation in the political movements, and his energy found fuller exercise in the prosecution of his original researches and in the exercise of his philological tastes. During the seven years that he remained here he kept up his study of Italian, Arabic, and acquired a knowledge of English. His scientific researches at Würzburg embraced the subjects of phthisis, tuberculosis, typhoid fever, cretinism, hydronephrosis, adipocere, echinococcus of the liver, amyloid degeneration of lymphatic glands, the corpuscles of bone, cartilage and connective tissue, and he thoroughly investigated the anatomy of the nails and the epidermis. While *Die medicinische Reform* was discontinued shortly after he went to Würzburg, the young professor, instead, edited a *Handbuch der speciellen Pathologie,* and, in connection with J. Vogel, issued a manual of general pathology.

It seems from a careful study of Virchow's career that it was at about this time that his observation of concrete facts became sufficiently extensive to justify him in venturing upon important generalizations; for the little manual issued in connection with Vogel contained many of the fundamental principles which a few years later were elaborated into his famous *Cellularpathologie,* in 1859. He had been recalled to Berlin in 1856, under circumstances that invested the incident with the characteristics of a triumph. The chair of general pathology had become vacant through the resignation of his former teacher Froriep; in all Germany there was none so able to fill it as the young democratic professor. He was sent for—but paused to consider. When his reply came it brought his acceptance, based, however, upon the condition that an institute for practical work should be founded. His terms were accepted, not only in this, but in other particulars, and he at once entered seriously upon what must be recognized as his more distinctive life work. The Museum at that time contained 1,500 preparations; at his eightieth birthday, as the result of his own individual labors, the number had increased to 23,000. In his work he was actuated by the view, expressed in his own words, that "the rôle of pathological anatomy as a dogmatic science is at an end; for each individual

law we must have the proof, clearly recognized and carrying personal conviction." He insisted that the whole of the then existing system must be abolished, and that a new philosophy, based upon observation and experiment, must take its place. This new pathology, he insisted, must come about gradually, and not as the mental product of individual enthusiasm. It must be achieved as the outcome of laborious research by many competent investigators, and, when thus evolved, and thus only, could it be accepted as the basis of scientific medicine.

The engrossing character of Virchow's labors at the Institute at this time, the absorbing enthusiasm involved in the promulgation of a new and revolutionizing philosophy, the exactions of editorial duty, all combined with the responsibilities of professorial work were not sufficient, however, completely to divert his attention from collateral and often apparently irrelevant studies, and from participation in the fierce political controversies that were then agitating the German people. William I. had ascended the Prussian throne in 1858. There was some hope of relief from the oppressive measures of his predecessors, and this very hope stimulated the activities of the Democrats or of the "Demagogen," as the party was appropriately designated by the conservatives. Virchow, notwithstanding his unpleasant experiences that had resulted from his banishment to Wurzburg, immediately identified himself with the cause of popular liberty. In this he was actuated by a profound contempt for the reigning house, a contempt which, on occasion found expression in his famous observation on heredity. "I know a family, a very exalted one," he was wont to say, "in which the grandfather had softening of the brain, the son hardening of the brain, and the grandson no brains at all," the reference being to the three Frederics, the immediate predecessors of the then reigning monarch. The work of the mere agitator, however, was not sufficient for one of Virchow's temperament, particularly to one who, after a previous election to the National Legislature, had been denied his seat on account of his youth. He was, in 1862, older by fifteen years, and, accordingly, offered himself as a candidate for the Prussian Chamber, to which he was duly elected. It was in the same year that Bismarck became Prime Minister, a coincidence which marked the beginning of an antagonism that continued throughout the political careers of the two men. Virchow speedily became the leader of the radical party, and by his advanced views and cogent reasoning, and by his courageous insistence upon them, he speedily earned for his policy the opprobrious designation of "Professorismus," applied by the Iron Chancellor. While these debates were going on, the

duties at the Institute, at the Charité and in the editorial office were not neglected, although there is ample testimony that Virchow was often tardy in keeping his appointments. The famous Schleswig-Holstein episode diverted for a time the attention of the Legislature from internal to external affairs, and culminated in the war with Denmark in 1865. In this war Bismarck against Virchow's opposition, used Austria as the cat with which to pull the chestnuts from the fire, and then, three years later, again over Virchow's opposition, he proceeded to kill the cat. As a result of this war with Austria in 1866, the Germanic Confederation of 1815 was terminated and the North German Confederation took its place. It may be premised that Virchow, who, with all his democracy, was always a Unionist and a Nationalist, deprecated this segregation of the Germanic people. It was in the course of this long sustained opposition to the policy of the Government, that he, as chairman of the finance committee, a position which he held for many years, succeeded in defeating an appropriation for naval purposes that had been demanded by Bismarck, who, thereupon, challenged his successful antagonist to mortal combat. Virchow, with no disposition, whatever, to give the herculean warrior an opportunity to exercise his professional skill, and with moral courage to stem the tide of sentiment in favor of duelling that still disgraces Germany—a courage vastly excelling mere physical bravery — declined the cartel, but continued his opposition. This opposition was carried along through the days of the Franco-Prussian War, but when the first shot had been fired, Virchow, always a patriot, and always the physician, took his son and joined the army, the two serving in the capacity of surgeons in the field. These men, father and son, did their full measure of duty, conspicuously upon the field of Metz, in alleviating the sufferings of their wounded compatriots. No sooner, however, had peace been concluded with the proclamation of William I. as Emperor of Versailles, than Virchow resumed his wonted activities in science, in literature, in politics at Berlin. It was then that probably for the first time in his political career he found himself *en rapport* with the leading features of Bismarck's policy, namely, the policy that involved the construction of the present German Empire. It may have been this particular fact, quite as much as a general appreciation of Virchow's worth, that prompted Bismarck, before his own retirement, under the present Emperor, to apologize publicly for many asperities which had characterized his previous attitude toward the great savant.

About this time the widened scientific view of Virchow, a view which had come to embrace the whole science of man as com-

prehended in the then slumbering science of Anthropology, began to be manifested in his contributions to literature. He was already accumulating facts which were to serve as the ground work of ethnology, yet, in spite of all this, acting in his capacity as a member of the Town Council, a position which he held for more than forty years, he was not oblivious to the fact that the sanitary condition of Berlin was deplorable. He, accordingly, became responsible for the establishment of those enormous hygienic reforms that have banished typhoid fever and other zymotic diseases from Berlin and that have rendered that city one of the most salubrious in the world. Archeology also was at this time engaging his attention, and in the midst of the preparation of his valuable work on the topography of Troy, he, in 1878, retired from active political life, only, however, to be elected, two years later, to the German Reichstag. In this body, however, he was always an interested spectator rather than an active participant, and never aspired to the office of party leadership. It is not to be assumed, however, from this that Virchow's intellectual activity was by any means at an end, or even upon the wane. The twenty years following his election to the Reichstag were among the most fruitful, intellectually, of his entire life. He amplified, in many particulars, his teachings of cellular pathology. He delved more deeply than ever into the hidden mysteries of ethnology, producing in 1882 his valuable work on "Old Trojan Graves and Skulls." At the very pinnacle of scientific fame, he kept himself *au courant* with the whole trend of scientific thought, delivering an address before an International Congress at Berlin, at Paris, at Moscow, or at Rome, laboring in an assemblage of scientists here, or in a hygienic congress there, or delivering a Croonian or a Huxley lecture in London. With his editorial labors always in hand, he still clung industriously to his old haunts in the Pathological Institute, in the Anthropological Museum, or in his ward at the Charité, for, be it remembered, Virchow was always a practical physician. It has been said of him that during these years he knew no such thing as vacation, in the ordinary sense of the word, for it was his habit rather to find recreation in a change of occupation, such, for instance, as visiting Asia Minor, and, pick in hand, to assist his friend Schliemann in his wonderful archeologic researches. In the midst of it all he was very much of a man on the human side—a little wiry man, but a little over five feet in stature—sprightly, congenial, loving and lovable. His domestic life has been described as ideal. The many Americans who were present at the Berlin meeting of the International Medical Congress, will recall his active and whole-souled partici-

pation in the festivities of that occasion. He was given a Festschrift on his seventieth birthday, and again on his eightieth, on which latter occasion, in particular, delegates were present from practically every country, and festivities were held simultaneously in practically every leading city of the world. On January 3, 1902, he sustained a fracture of the neck of the femur, by falling from a tram car. He died September 5, 1902, mourned by the civilized world. The municipality of Berlin, which he had faithfully and efficiently served as a Councilor for so many years, accorded him the distinction of a public funeral, which in the midst of universal mourning, was participated in by many officials from the political and scientific world.

This, then, was the man upon whose work we are called, at this hour, to pronounce a formal appreciation. It is rare, indeed, that the occasion arises to attempt in even a desultory way, the estimation of a career that has resulted in the establishment of two distinct, although correlated sciences, and in the substantial advancement of human liberty. It would be quite out of the question in an address such as this, to attempt a resumé of his doctrines in pathology, a mere enumeration of his contributions to which would involve the employment of more than twice as many words as I shall employ in your hearing. We may, however, arrive at some estimate of his work in this great department by pausing for a moment to consider the state of medical science, or more particularly, the conceptions of disease, that obtained in Germany when Virchow was made the successor of Froriep at Berlin. It is true that Rokitansky had introduced many of the revolutionizing doctrines of Bichat at Vienna, but even Rokitansky was busying himself to an important extent in promulgating the purely dogmatic doctrines of crases. Oken, at Munich, was indulging in the glittering generality that life is the self-generation of individualized elements, that the principle of life is galvanism, and that vital force is galvanic polarity. Of him Agassiz declared that he constructed the entire universe out of his brain. Dollinger of Würzburg, the father of the great theologian, belonged to the same speculative school, which a historian has designated as the "Romanticist or Teutomaniacs." At Berlin Schönlein, who had been one of Virchow's teachers, and was yet his colleague, and who represented what was designated as the Natural History School, taught that disease was an entity, a sort of parasite sojourning temporarily in the body, just as Paracelsus had once spoken of "a microcosm within a microcosm." Schönlein, more specifically, looked upon disease as a sort of equivocal infusoria, the existence of which he logically predicated, but never, of course, physically demonstrated. These

infusoria, the existence of which were thus gratuitously assumed, were easily enough imagined to consist of genera and species, each producing different clinical phenomena—a sort of empirical prophecy of the germ theory which has since played so important a rôle in medical philosophy. It was against such theoretic doctrines, then dominant, that Virchow brought the evidence of the microscope and the revelations of the mortuary. He began in the truly scientific manner, which consists always, first in the observation of concrete facts, next in their classification, and third in their ultimate generalization. His labors at Würzburg, supplemented by those conducted under more favorable auspices after his return to Berlin, enabled him to announce, as the seminal doctrine of his philosophy—and I employ the words he subsequently used at the Fiftieth Congress of Naturalists and Physicians, namely, that the new science was based "chiefly on the recognition of the fact that the cell is actually the ultimate, proper morphological element of every vital manifestation—*omnis cellular e cellular* — and that we must not remove the proper action beyond the cell." In the early elaboration of this doctrine, taking up the work where Schwann and Schleiden had left it, he early proclaimed the importance of the nucleus to the maintenance and multiplication of the cell, and emphasized the fact that tissue growth implies cell multiplication, while the contents of the cell, and even the material deposited outside of it, are of controlling importance to function. He taught, furthermore, and as a necessary corollary of the preceding postulates, that tissues vary in function according as they vary in cellular construction. He insisted upon the existence of an intercellular tubular system, that supplemented the recognized circulatory systems in the work of ultimate nutrition. As a result of his investigations of the circulatory apparatus and of the blood, he taught that the walls of the blood vessels were impervious and argued that blood, or even the nutrient elements of the blood could not escape from them without rupture of the walls, which rupture was, however, rarely, if ever, demonstrable. It would seem that in this doctrine, which I believe is as near an approach to empirical dogmaticism as could be found in all his teachings, Virchow laid a logical foundation for the new doctrine of Osmosis that to-day promises to take both physiology and pathology largely into the realm of physics. He directed his arguments specifically against the then prevailing humoral pathology by insisting that the blood itself is not the proper and original cause of dyscrasiæ, as taught even by Rokitansky, but that instead these dyscrasiæ have their origin rather in a disturbed metabolism, the toxic products of which are merely carried in the

blood. His laborious study of many phases of blood changes comprise the basis of our present accurate conception of the pathology of the circulatory medium. In his study of inflammation, in the description of which he insisted that disturbed function should be added to heat, pain, redness and swelling, as one of the cardinal indicæ of the phenomena, he gave an accurate conception of the actual changes. His elaborate investigations of the nervous system resulted in the promulgation of doctrines whose parentage in the works of Brown and Haller is recognizable. His work on tumors, a distinct application of the cellular doctrine, stands to-day as the fundamental classic of the subject. His investigation of tuberculosis resulted first in his classification of the diseases into neoplastic and inflammatory forms, but latterly he recognized the bacillary forms. It would be impossible, however, as I have before stated, to give even an accurate resumé of this extensive philosophy, the application of which to the entire phenomena of disease must stand as his crowning achievement. It is interesting to hear him recount, as he did in a lecture delivered in London, during the last years of his life, the general summarization of his work in the statement that "the law of continuity of animal development is, therefore, identical with the law of heredity, and this I was now able to apply to the whole field of pathological new formation." And it was especially interesting, in view of the ideas against which he had to contend, to hear him add with pardonable exultation: "I blocked forever the last loop-hole of the opponents, the doctrine of specific pathological cells from which types and ancestors were not forthcoming in normal life." The doctrines which he had thus established, and to which he thus alluded, became, early in their history, the actuating principles of the "Berlin School," which sooner or later embraced the names of Leyden, von Rechlinghausen, Cohnheim, Waldeyer, Hoppe-Seyler, Kuhne, Rindfleisch, Klebbs, Liebrich, Frederic, in Germany; Felix Simon, in England; and, conspicuously W. H. Welch in the United States. The principles taught by this school are, by common consent, those upon which modern surgery and rational therapy alike are placed.

The position that must be accorded to this doctrine in the light of further revelation of fundamental law, cannot be foretold. Nothing could be further from the purpose of Virchow himself, than the assumption that his doctrine was the all-truth. He viewed with great conservatism the doctrine of the infectiousness of disease based upon observations that were made possible only by a later perfection in optics and by later advancements in the technique of biologic research. The most that can be

said of the relation of the germ theory of disease to that of cellular pathology is, that without invalidating the important conclusions embraced in the latter, it left Virchow's recorded observations unimpaired and undisputed. The new doctrine of the ions, involving the principle of Osmosis, may bring other and important supplementary facts which shall serve to show that the discoveries of Virchow comprised in the aggregate a single, but important link in the evolving chain of science.

The next phase of Virchow's character as a scientist relates to his work in the department of anthropology. This, the science of man in its broadest conception, can scarcely be said to have had more than a mere beginning before Virchow, beginning with his work in biology, was led into it by the widening circle of associated ideas. It may be said, indeed, no valuable contributions were made to the subject during the first half of the nineteenth century. Blumenbach, Göttingen, had made his famous collection of skulls,—his "Golgotha" as he called it,—which was the basis of his own investigations, and which may be said to have been the starting point of systematic anthropological study. About the same time, that is the last years of the eighteenth and the first years of the nineteenth century, von Sommering, of Frankfurt, studied the eyes, not only with reference to their anatomical detail, but with reference to their ethnic significance, while Camper, of Holland, made a careful study of the facial angles. This was practically all that was done with the subject, until Darwin issued his "Origin of Species," in 1859; his "Descent of Man," his first contribution to the subject of ethnology, did not appear until 1872. Long before the latter date, however, Virchow had taken up the subject at two points of contact. The first point of contact was developed out of his philosophy of cell genesis, the doctrine that all cells are derived from pre-existing cells, which he promulgated in 1859, and which brought up, as a natural corollary the question of variation of type. His antagonists—the believers in special creations—seized eagerly upon this declaration as a refutation of the then rapidly growing materialistic philosophy, and as a vindication of their own ontologic dogmas. If the cell is the vital unit, as Virchow declares, and if the individual is but the sum of cells, they urged then, variation in the individual cannot only occur as the results and commensurately with the variation in the constituent cells; if, they added, like cells always beget like cells, as Virchow declares, then the individual, the sum of cells, cannot vary from his cellular type; and, finally, they insisted, if all the cells in the individual have been derived through the generations from cells of the same type, then the original cells,

at the beginning of things, must have been the products of a special and miraculous creative act. Unfortunately, however, for this specious logic, Virchow taught, in effect, that like cells begat like cells, only, however, under like circumstances, and that, as the circumstances vary, so does the cell type vary. This is, indeed, the point of departure from the standard of health, the very beginning of pathological phenomena. As a matter of fact, Virchow simply declined to discuss the origin of species until sufficient evidence to justify him in doing so could be derived from a careful search of the tissues. He recognized the mutability of the cells, and realizing, logically, that variations in type must begin in these vital units, he, without denying the truthfulness, or affirming the falsity of Darwin's hypothesis, simply awaited the demonstration of the actual changes within the cell. It is an interesting fact and one bearing testimony to Virchow's scientific acumen, that this very variation was reduced to a physical demonstration in 1900 by Professor Guyer, of the University of Cincinnati, whose investigations are recorded in his valuable contribution on "Hybridism and the Germ-Cell." It is also of striking interest, at this time, and one bearing testimony to the reliability of Virchow's deduction, not only that these observations of Guyer's, but that Mendel's Law promulgated through an obscure periodical at Brunn, Austria, in 1865, seemed to cover the entire point. This Law of Mendel's, or, as I believe we should now call it, the Mendel-Guyer Law, is in effect that, as the result of definite and demonstrable changes in the germ cell, the second and later generations of a hybrid possess every possible combination of apparent characters, and that each combination appears in a definite proportion of the individuals, the whole reduced to the terms of a definite equation. This law, revealed by observations in both the animal and vegetable world, seems to be one of general applicability, and one that is calculated to invest the conclusions of Virchow with an increased value.

The next point at which Virchow was brought in contact with the general problem of anthropology, or more particularly, that of ethnology, grew out of his studies of cretinism and of the causes of variations in the growth of the skull. It was precisely this study of the pathologic phases of craniology that enabled him to detect morbid changes in the celebrated Neanderthal skull, which, with its protruding supraorbital ridges, its low forehead, and its small cranial capacity, even the scientific world was too disposed to accept as the normal index of a racial type that had long since passed away. Virchow further insisted that, even if it were normal, the existence of a single skull were not sufficient evidence upon which to predicate the

existence of an entire race, and that conclusions should be withheld until further evidence was secured. It was this cautious utterance, thoroughly characteristic of Virchow, that gave the theologic polemics another opportunity falsely to proclaim that he was an antagonist of the doctrine of descent, as promulgated by Darwin. It seems that the utterance, seized upon for this particular misrepresentation, occurred in an address delivered in 1877, before the German naturalists and physicians, and was to the effect that the hypothesis of Darwin ought not hastily to be given the force of law—that it ought not to be placed in the category of law—without first waiting to gather and accumulate all relevant facts. It was just this scientific discrimination between hypothesis and law, and just this conservative tendency in the consideration of demonstrated facts, and in the formulation of conclusions based upon them, that gave to the judgment of Virchow the greatest possible weight in the scientific world. And it was this very weight which he, himself, as late as 1900, with true scientific spirit, was disposed to deprecate; for he had spent his life in dethroning the power of personal influence, and in establishing the regnancy of demonstrated truth.

His work in anthropology, however, considered from its positive side, was very great. He was always an organizer—a valuable weakness in a man of brains,—and it was by this means that much of his work was brought to its full fruition. He organized, or at least assisted in the organization of the German Anthropological Society, and the *Berliner Gesellschaft für Anthropologie, Ethnologie und Urgeschichte;* he helped to found the *Museum für Volkstrachten* and the almost invaluable *Archiv für Anthropologie.* He, with his colleagues, gave serious study to the physical characteristics of the early Germans. This was supplemented by statistical investigation of the present distribution of the color of skin, eyes, hair in Germany, the whole being reduced to cartographic representation. His descriptions of American crania, based upon Morton's great work, opened that mine of information to German thought. He was the friend and promoter of Schliemann, in whose archeological explorations he was at times a personal participant; he recorded the results of these labors in two books, " Contributions to the Topography of Troy," and "Old Trojan Graves and Skulls," each of which is recognized as a valuable contribution to the subject. Extensive, however, as were the researches, and important as were his recorded observations, it does not appear that he considered either of them sufficiently extensive to warrant him in arriving at important general conclusions. He felt justified, however, in saying, as he did say, that physical types do not vary with variations in language and culture, and that different types may blend in

the formation of a homogeneous people. This lesson was taught him by a study of the racial types in Germany, and is of extreme interest to us in the United States, where, at this moment, we are in the midst of the greatest ethnic experiment in the history of the human race. In viewing the entire scope of Virchow's labors in anthropology, it must be concluded that he did not carry them to the point of even relative finality that he did his labors in pathology; his researches, his discoveries in ethnology must be recognized as fundamental, their true significance remaining to be interpreted in the light of rapidly accumulating evidence. It is sufficient, however, for the perpetuity of his fame, that, by common consent, he is recognized as the veritable founder of this new science which promises so much for the interpretation of the racial types of men.

The third side of this great character was the human side, manifesting itself not alone as a husband and father, but conspicuously as a citizen. He early showed that the prevalent opinion that to be highly intelligent on one subject it is necessary to be correspondingly stupid on all other subjects is but a vulgar notion; and he speedily demonstrated that the view point of the physician is eminently calculated to afford an intelligent insight into social, economic and political conditions. I must, however, leave it to the political historian and to the public economist to tell what good has been accomplished in Germany in the last fifty years by the liberal movement, a movement that, for many decades enjoyed the distinction of Virchow's leadership. A few things are certain: the hated Carlsbad Decrees could not be re-enacted in Germany to-day; there is a greater freedom of thought, and, what is more important, of expression in German universities than ever before; the offense of Lèse Majestè, strange sounding to Rupublican ears, has a less severe meaning in Germany than it had fifty years ago; and it is equally certain that, for the first time in history, the entire Vaterland has a reasonably liberal constitution, wrested from the tyranny of absolutism—a condition that leads to the hope that the German people may some time enjoy the same beneficent government that to-day blesses the Great Republic. In the achievement of these results it cannot be denied that Virchow played a leading and an honored part.

What, then, are we to say in final review of this great man? His figure is that of a colossus, and it will require the prospective afforded by receding years to measure its relative height. Some things, however, we can now tell. He inherited honest blood; he responded in the fullest and in the best sense to the formative influences with which his early life was surrounded; he had the independence to defy personal dictum and to give

allegiance only to demonstrated truth; he had the intelligence to discern and the human sympathy to appreciate that human happiness depended first and chiefly upon a knowledge of the laws underlying and governing human existence; he worked on independent lines and revealed laws of disease previously hidden; he, by his observations and deductions, and by the elaboration of rational methods, laid the foundation of modern medicine; he established the study of racial man as a science; he fought the battle for human liberty and won for others the boon that he had always arrogated to himself; he added years to the generation of man, brought happiness to his kind; finally let it be recorded, that, above all, he lived faithful to his ideals—and the greatest of these was Truth.

REMARKS BY Dr. A. JACOBI, of New York: Mr. President: I rise to make a motion, and in doing so I wish to say that perhaps never before in my life have I risen with greater diffidence, and with less courage than at the present time. I am still under the influence of this eulogy, or rather this address, for it is not a eulogy but a simple statement of the truth. Every word that has been spoken is true, and I have been deeply impressed with it.

I knew Virchow well and had the honor of being included among his friends. He was everything to everybody; he was everything to his country. He was a particularly warm friend of America, not that he paid more attention to American science than to that of other countries, but he loved this country because he knew that it had done a great deal more in certain things than any other country in the world. I refer you to what he said repeatedly about the lessons America had given to all mankind, particularly with regard to its achievements before and after the Civil War. No one ever lived who more fully appreciated the reduction of mortality in war than that effected in America. Whenever an American approached him he always had in mind the fact that he, and all mankind, owed much to America, and usually American students and physicians were preferred by him.

He never lost sight of the claims of practical medicine, of therapeutics. On the very last page of his Cellular Pathology, which appeared more than forty years ago, he told his audience that he hoped in these twenty lectures he had given them something in this cellular pathology which they could utilize in their practice. He himself was always loath to give up his practice in the Charity Hospital, and it was only in his last years that he was compelled to do this because the practice was claimed by others and he had too many other things to do.

The only thing that is left for me to say is that I firmly believe that this address will stand forever amongst the foremost appreciations of Virchow's greatness. The least we can do is to say that our thanks are due to the gentleman who came from the far West to deliver this address, and that we herewith tender them. That is my motion, Mr. President, that we extend a vote of thanks to the distinguished gentleman who has delivered this remarkable oration upon the life and character of Rudolph Virchow. The motion was carried unanimously.

COMMUNICATIONS

I. SOME SCIENTIFIC AND PRACTICAL ASPECTS OF VACCINATION

By P. H. BRYCE, M. A., M. D.

SECRETARY OF THE PROVINCIAL BOARD OF HEALTH OF ONTARIO, TORONTO, CANADA.

Mr. President and Gentlemen of the Medical Society of the State of New York:

I HAVE to express my appreciation of the favor conferred upon me through the invitation to address your Society on a subject which, however old and hackneyed, becomes in view of its present importance one of extreme interest.

After a hundred years since Edward Jenner's discovery had been accepted in Europe, and by some of the more eminent physicians of America, we find a disease, which in the eighteenth, and preceding centuries had its victims indifferently in the hovel and the palace, so little prevalent, that until within the last three years the majority of living physicians in America and Canada had never seen a case of smallpox; and, as it has happened, the great number of cases which have been seen on this continent recently have been so mild, that we have to go back to the days of Sydenham in the seventeenth and of Van Swieten in the eighteenth century to find a parallel for this anomalous type of the disease. It therefore has not been unnatural that to those inexperienced in the appearance of the disease, and even to those familiar with the type of the disease as it has appeared when introduced from Europe, much doubt has arisen as to whether or not the disease, which was brought from Cuba in 1898, and spread so widely over the Southern, Western and Central States and Canada, has been true variola.

Our supreme test of vaccination however, has enabled us to prove conclusively its nature, since the disease has been combatted as successfully by that wonderful discovery of Dr. Edward Jenner, as the numerous outbreaks were which from time to time had previously appeared since 1800.

What Jenner taught in brief was that by vaccination we introduce by inoculation a disease which causes a vesicle of a par-

ticular character on the teats of a cow, or on the tender skin of the belly of a calf, the virus of which when inoculated into a person produces a vesicle of a similar character.

We now know, what Jenner and other experimenters of his time believed from their experiments in variolation or inoculation with smallpox virus, that a similar vesicle can be produced through inoculating several calves in a series with virus from a smallpox patient, and that this smallpox virus thus modified produces in other calves, in man, monkeys, and in guinea pigs, a vaccine which protects against smallpox completely, in practically all cases for ten years, in a large percentage for twenty years, and which, though decreasing, continues to protect against the severity of an attack of smallpox to a large degree throughout life. In the absence of any knowledge of the germ theory of disease, it is natural that Dr. Jenner and his associates should not have arrived at any clear idea of how this protection was produced, further than that it was the same as that caused by other eruptive maladies, whether in man or animals. Not until Pasteur's discovery of the germs of anthrax and chicken cholera and his success in producing immunity by the cultivation of the micro-organisms of these diseases, did it become possible to formulate any theory as to how the protection by inoculation with the germs of a disease was brought about.

Without discussing the modern experimental evidence regarding immunity, we have only to realize the organism of any bacterial disease as a simple cell, having its environment within the body, subject to variations, just as in nutrient media, and multiplying and completing its life-cycle in the body, obtaining its pabulum from the fluid tissues and their contained cells, and producing substances peculiar to itself, acting upon and being re-acted upon by the normal tissue-cells, in order to understand that its pabulum may be exhausted as in a culture medium, that its products may become auto-toxic, and that they may stimulate in the tissue-cells of the body the production of compounds inimical to the further development of the micro-organism of the specific infection.

We may, therefore, very properly conclude that as the bacillus of diphtheria grown outside the body produces its toxin, which introduced into horses produces the antitoxin, whose quality and strength can be measured, so the variolous or vaccinal germ produces its toxin, which stimulates the tissue cells to produce their antitoxin.

That such is actually the case, has been experimentally proved, especially by Beclère and Chambon of Paris, who have shown that active vaccine lymph may be neutralized by adding to it in

test-tubes the serum from vaccinated heifers, or from men or monkeys recently having had variola. We have every reason therefore to conclude that variola is a bacterial disease, and that Copeman's, Klein's and others' claims that they have isolated the micro-organism of smallpox or vaccinia, are founded upon fact.

The progress of vaccinal immunity in calves has further been admirably illustrated by the experiments of Beclère and Chambon, in which subcutaneous injections of active glycerinated lymph were made; and subsequently epidermal inoculations were made on successive days from the third to the seventh. The effects on the vaccine vesicles when the scarifications were made after the fourth day, were:—(a) vesicles appearing sooner than in normal vaccination; (b) vesicles modified in external appearance, rapidly arrested or aborted in development; (c) lymph having little or no virulence when taken from vesicles after the fourth day. Such in brief is the basis upon which the immunity caused by vaccination rests; and it must be satisfactory to all who have followed the marvelous results of the biological studies of infectious diseases, carried on during the past quarter of a century.

It is, however, a remarkable fact that while the practice of protective and curative inoculations in the instance of diphtheria has been generally accepted both by the profession and the public there has nevertheless grown up during the very period in which the experiments which form the ground-work of all our theories of immunity have been carried out, an opposition both to the theory and practice of vaccination against smallpox, which even in conservative England, which claims the honor of the great discovery of Dr. Jenner, resulted in 1896 in the introduction of the conscience clause in the compulsory Vaccination Act.

Wherein, then, lies the origin of this opposition? Primarily, I believe, it lies in the simple fact that vaccination laws are compulsory. We have in fact no other law compelling persons to subject themselves to inoculation with a disease, at a time when they are in perfect health, and when as a matter of fact no cases of the disease may exist in their community, or indeed in their country.

In the second place, the fact exists that in an occasional case unfortunate results have followed the operation, giving some reason for the objections which have been raised.

What moral grounds then can we have for insistence upon the necessity for such legislation; and, if such can be shown to have a basis in reason, what are the logical deductions to be drawn as to the moral duty laid upon the State which institutes such legislation?

With regard to the first question we have several answers:—

First. That vaccination has during a century been the means of reducing the mortality from a disease, which previously caused one-tenth of all deaths in European countries, to the lowest of any of the infectious diseases which we have to combat in temperate climates.

Second. That the theory of the immunity caused by it is based upon experimental evidence, which has completely revolutionized the practice of medicine, and produced results in the instance of such diseases as anthrax, rinderpest, plague and diphtheria, which are comparable to that of vaccination itself.

Third. That inasmuch as the infectiveness of smallpox is incomparably greater than that of any of these diseases, experience in every country has shown that, while sanitation, isolation and disinfection play important parts in the work of prevention, even the most complete sanitary organization has utterly failed again and again to eradicate the disease from a community without vaccination.

Fourth. That we have the marvelous fact that vaccination is adequate to protect completely against the disease after exposure has taken place, even up to the fourth day, and of reducing the severity of the disease to a non-fatal issue in almost every instance where vaccination is concurrent with the smallpox.

If then we have such potent reasons for persisting in our demands for compulsory vaccination, we must be prepared to accept the fullest responsibility for the position taken, which must be that, if we insist on compulsion, we shall not through indifference or neglect allow anything to exist or take place by which any element of danger can enter into the results of the operation.

I am quite prepared, gentlemen, to admit that while any serious results, which have ever been shown to follow the operation, are in practice infinitesimal compared with the total number of operations, yet the secondary effects of the operation, at times, from the ethical standpoint, have been such as to clearly inculpate the producer, the operator, the patient, or two, or indeed all three together.

As regards the producer we must realize that, while bovine vaccine has been produced and used largely during the past twenty years in America, the methods adopted, being in the hands only of private producers, would not be likely to be improved beyond the scientific knowledge of the biological theories explaining the mode of its production. Not until bacteriology had made known to us the part played by staphylococci and streptococci in pyæmia and septicæmia, could we understand

why the secondary infections were unnecessary and avoidable complications of vaccination, recognized however as to their existence even by Jenner, who said, "That the most material indisposition, or at least that which is felt most sensibly, *does not come primarily from the first action of the* virus on the constitution, but that it often comes on *if the pustules be left to chance,* as a secondary disease." Hence it was not uncommon up to quite recent years for lymph to be taken from vesicles on a second or even a third day, and for clamps to be used for extracting the largest amount of lymph possible from the vaccinifer. Within the last ten years, however, with the experimental work of Blaxall, Copeman and others, all this has changed; and to-day we have producers everywhere supplying or endeavoring to supply, a vaccine free from extraneous organisms. As usual, the very virtue of the method has become in some instances a defect, and it is found that at times the activity of the virus itself has disappeared. New producers have entered the field, widespread outbreaks have created unexpected demands for vaccine; and between inexperience and commercial necessities, the practice of vaccination has been injured by lymph at times of excessive virulence, and oftener by that having no protective value. It is, therefore, apparent that until all vaccine sent to the operator has been tested, and indeed standardized as diphtheria antitoxin, by experiments on persons and animals, we must feel that the ethical demands of the situation have not altogether been met.

That it would make lymph more costly, can be no valid reason for its not being done, and no State with a compulsory law can evade the responsibility for neglecting to demand of producers that all vaccine supplied be tested or, failing to secure this, must supply adequate facilities for its production by qualified State officers. When we turn to the operator or public or private vaccinator, we find that, while the State licenses medical practitioners, there seems to have been everywhere on this continent a growing neglect on the part of Medical Colleges to either teach the theory or illustrate the practice of vaccination. We find lymph which has been stored for weeks in a drug store. used by the practitioner, as if it were an inert mineral drug, incapable of change; and while, in a surgical operation of another kind, the practitioner may carry out aseptic precautions to an almost absurd extent, he will invade the uncleansed epidermis with perchance an unclean scalpel, and after scarification leave the unprotected wound to its fate, with an unthinking disregard as to whether its course may be normal, or a dangerous secondary infection supervene. In all this the personal elements, as regards

scientific knowledge and personal responsibility from the ethical standpoint, are points which perhaps it would be too much to make the State responsible for; but if the credit of vaccination is to be lessened or a single person injured by the operation, then it is clearly the duty of the State to allow such compulsory work to be performed only by responsible trained public vaccinators, and to establish heavy penalties, as is done in Germany, for any unqualified person performing the operation, or for proved carelessness on the part of the public vaccinator.

It is apparent that to institute such a system, legislation of a kind similar to that of England and Germany, would be essential. In England, and especially in Germany, such compulsory vaccination is accompanied by provisions for the production of vaccine by State establishments, where the responsibility for the production of tested lymph properly rests, and whence lymph is supplied to qualified public vaccinators appointed by the municipalities. The system further provides for the regular and systematic vaccination of infants and of revaccinations on entrance to the schools and to the army and navy. Such desiderata are making themselves felt more and more on this continent, where increasing urban populations and the facilities for the transmission of infection through the extending travel by railways are equally becoming greater.

With regard to the individual responsibility for unfortunate results of the operation, little need be said. If the vaccinator does not warn the patient of the precautions to be taken, we may expect that the latter, too frequently, through ignorance, will be lacking in a knowledge of the care necessary to protect himself. He ought to be taught to know that he is inoculated with a disease, and that for the short time required he must consider himself a patient. In the 1900 Report of the Medical officer in charge of the vaccinations at the National Vaccine establishment, London, we are informed that out of 1892 primary vaccinations, eleven on subsequent inspection showed some abnormal cause, most of which consisted of " sore arm " caused by *domestic maltreatment*.

In view of the distribution by producers of lymph, through the journals of this country and through advertising circulars within the past three years, of statements regarding vaccination, which at times have been at variance with the authoritative teaching on the subject, it may not be ill-timed to refer briefly to the question of what constitutes a normal lymph and a normal vaccination.

It must be remembered that for years we have looked upon a good vaccine as one, which by its more rapid evolution, will

within eight days have developed so complete a vesicle that it will serve to protect a person inoculated up to the fourth day after an exposure to smallpox, the incubation period of which is from twelve to fourteen days. We learn from report after report of the National Vaccine establishments in England, Germany and France, that the vesicle on the calf is mature within ninety-six to 120 hours after inoculation. So fixed for many years was the period of maturation of the vesicles in man, that the compulsory laws of England required all children to be brought on the eighth day for examination of the pock. According to Copeman of the London National Establishment, glycerinated as well as crude lymph, if normal, will have produced by the seventh day a vesicle five or six m.m. in breadth with a glistening, translucent margin of a nacreous or pearly appearance, with the pale-red areola, the rest of the surface presenting a more opalescent bluish-white appearance; while the patient suffers from malaise, with some inflammatory fever, and involvement of the axillary glands. From this pearly margin our old teachers took the lymph on the eighth day for arm to arm vaccination. With this picture so constant for a hundred years, it is astonishing that we should recently have had new teachers informing us that glycerinated lymph produced normal vaccination, when a vesicle had matured on the twelfth day, without, as many of us know, presenting the pearly border and characteristic vesicle which we had been taught to look for. It is apparent that if we are to accept this new teaching, the protective inoculation after an exposure to smallpox, would become impossible. Fortunately this new doctrine has been short-lived; and personally, I have been able to demonstrate even to the satisfaction of producers, that a normal lymph, even though it may have had to overcome some slight immunity, was able to produce on the eighth day, in a patient so vaccinated, a vesicle of quite typical character. Such lymphs, therefore, we have unfortunately had good reason to know, have not only not protected against another vaccine, but have not protected against the mild type of smallpox which has so generally prevailed.

It has hence become essential to the maintenance of the credit of vaccination, that public officials, as well as the profession, should not only have perfectly clear ideas as to what a normal vaccination is, but that they further be placed in such a position in their several States and cities as to insist upon the use of only such vaccines as will produce normal results and effective immunity.

One of the results of these new theories has been to cast discredit on the very great advances which have been made

through the introduction of aseptic glycerinated lymph. Articles everywhere have been appearing in the medical press condemning unsparingly glycerinated lymph; and from the standpoint of the results above indicated, with some reason. But it is apparent that when we can obtain statistical results collected from hundreds of public vaccinators under an organized system, where as in England in the year ending March 31st, 1902, lymph for 974,595 vaccinations had been sent out from the National Vaccine Establishment; and where for the quarter ending Dec. 31st, 1901, 261,044 vaccinations showed a success of 97.9 per cent. and an insertion success of 93 per cent., we have a basis of fact which should wholly disabuse our minds of the idea that glycerinated lymph is a failure.

The actual figures published are:—

	Cases	Case success per cent.	Insertion success per cent.
Primary vaccinations	126,209	98.6	94.0
Re-vaccinations	134,835	97.2	92.0

Another theory which has likewise had its rise in these modern days of "sweetness and light," is that one scarification is sufficient for protective purposes. Contrary perhaps, to what the theory of immunization might lead us to conclude, viz,:—*that so long as the system becomes inoculated, it does not make any difference how the lymph is introduced, whether by one or five scarifications,* we have the statistics of more than half a century, proving absolutely that it does make a difference whether much or little lymph be introduced. We have long been accustomed to say that a well-pitted person will never take smallpox again; and in practice this is true. We say that a child, which has suffered from a severe type of measles or scarlet fever, is absolutely immune against another attack; and in practice this is true. We surely then are justified in saying that, just as the very slight protection produced by a twelfth day vaccine has not established an immunity even for a month against an active vaccine, so a single scarification does not allow the same absorption at one time as several would of a virus whose activity is to overcome the vital resistance of the tissue cells; and will not therefore call up in the same degree the vital energy of these cells to produce those anti-bodies, whatever their nature, upon which we now depend to explain the immunity.

The Table of Marson, surgeon to the London Smallpox Hospital, from 1836-67, of 13,755 cases of smallpox, seems conclusive on this point.

13,755 cases of smallpox, classified according to the vaccination found on each	Percentage of Deaths 1835-1851 3,094 Cases	1852-1867 10,061 Cases
Stated to have been vaccinated, but no cicatrix	21.7%	39.4%
Having one vaccination cicatrix	7.6%	13.8%
Having two vaccination cicatrices	4.3%	7.7%
Having three vaccination cicatrices	1.8%	3.0%
Having four vaccination cicatrices	0.7%	0.9%
Unvaccinated cases	35.5%	34.9%

Such facts would seem to be conclusive, and fortunately they do coincide with most of the knowledge we have concerning this wonderful fact of immunity against eruptive diseases, either through contagion or experimental inoculation.

It is apparent, gentlemen, that the few practical questions I have touched upon only serve to illustrate to what lengths the subject would lead us, if we attempted to do it justice.

It is, however, the practical side to which public officers of health have constantly to direct their attention; and it is apparent that we on this continent, with our democratic methods, have hitherto been greatly limited in our powers to either control the quality of vaccines, which have been supplied, or produced in State establishments, products which would fulfil the requirements.

Within the last year, however, official opinion has been crystallized, and its conclusions briefly stated would seem to be:—

First. That official supervision of the products of Vaccine Establishments by either State or Federal officers is imperatively demanded.

Second. Or that the production in State or Federal Vaccine Establishments of adequate supplies of vaccine of established quality to be sent out free or at cost to municipalities, has become a necessity.

It is not necessary here to enter into a discussion of which scheme is preferable; since, while Government production in Germany, England, etc., has proved a success, it cannot be forgotten that in some respects our democratic methods do not always conduce to that permanency of the Civil service and freedom from political complications, which are necessary to efficient performance of scientific work.

On the other hand the unlimited capital and keen competition of the large commercial houses supply in many ways facilities for the production of standard articles; and for their maintenance at a high standard of excellence, which perhaps few individual States could attain to.

Personally, I am convinced, however, that no matter which scheme is adopted, there is demanded before anything else, adequate legislation whereby from year to year, qualified public vaccinators must be appointed in every municipality, who shall be empowered to vaccinate systematically, and register all children, born in any year; and that through the responsibility laid upon them, and the routine methods established, such men will become experts in the art, and will gradually obtain such a store of information as will prevent the use of any except standard vaccine, and by the care exercised, be able to anticipate and prevent those accidents, which we have already referred to as seriously injuring the credit of this greatest triumph of modern medicine.

II. SYMPOSIUM ON ARTERIO-SCLEROSIS

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1. The Early Diagnosis and Symptoms of Arterio-Sclerosis

By DeLANCEY ROCHESTER, M. D.
BUFFALO

The recognition of an advanced case of arterio-sclerosis, with its thick-walled, inelastic vessels, hypertrophied heart and other organs presenting varying grades of impairment in nutrition and function is as easy as the induction of improvement in such a case is difficult. It therefore behooves us to so study our cases that we may recognize the condition in time to check its progress. In almost no other class of cases—except the distinctly infectious—does etiology have such an important bearing from a diagnostic standpoint. Therefore a most careful investigation of the occupation and customary mode of life of the individual, as well as of his heredity and previous diseases, is in order.

Etiologically cases may be divided into those due to strain of occupation, those due to poisons introduced from without, into the economy, and those due to poisons arising within the individual caused by faulty metabolism, either anabolic or katabolic. Into any of these classes heredity may enter as an etiologic factor of considerable importance. Into almost all cases two or more of these etiological factors enter.

The occupations that produce great muscular strain are those in which heavy lifting, carrying, hoisting, etc., occur.

The poisons introduced from without are syphilis and other infections, lead and alcohol. Those arising from within are

varied in character, some absorbed from the gastro-intestinal tract, arising there from poor quality of food or impaired digestive power of the individual, some due to impairment of the excreting function of skin, bowels or kidneys, and some due to faulty cell metabolism resulting from deficient oxygenation. Gout and lithæmia probably come under this heading. These poisons, according to Dr. Sansom, act either directly upon the internal coat of the vessel or in the lymphatics which are so abundant around the external coat, or in the lymphatics in the fibrous tissue of the various organs. These poisons show the result of their irritation by fibrosis, usually most pronounced in the subendothelial layer of the intima, preceded by degenerative changes in the media and followed by extension of the fibrosis into the media and externa. The symptoms are so varied, according to the cause and the organ or organs in which the disease is most pronounced, that a clinical picture applicable to all cases cannot be made.

The symptoms may be classified under the heading of (*a*) cardio-vascular-hæmic symptoms, including those preceding and accompanying gangrene; (*b*) cerebral symptoms; (*c*) renal symptoms; (*d*) pulmonary symptoms and (*e*) symptoms referable to the gastro-intestinal system.

Beginning with the last I will briefly review the symptoms in each class and conclude by a statement of the essential points in early diagnosis, leaving the details in each class to be taken up by the succeeding speakers. The gastro-intestinal symptoms referable to arterio-sclerosis are chiefly those due to interference with functions consequent upon the diminution in blood supply to the secreting apparatus and to the musculature of the viscera. Among these symptoms stand prominently forward constipation and indigestion, both intestinal and gastric, in some cases one portion of the digestive tract showing the greatest disturbance; in others, another portion. Distension of the stomach or bowels or both, flatus and eructations are not uncommon.

In some cases the pulmonary symptoms are so pronounced that one writer describes a clinical group that he calls that of "Spasmodic Asthma and Pulmonary Affections." The following case is a good illustration of this class of cases.

J. H. B., age twenty-eight years; American; carpenter. Family history good. Personal history: habits, tobacco in excess, liquor moderate. Diseases: gonorrhœa and probably syphilis in 1897; injury with severe secondary hemorrhages in 1899; rheumatism, possibly gonorrhœal arthritis in 1900. No other diseases. Early in 1902, after hard work and exposure, he had an asthmatic attack of considerable severity; he has had several since, all accompanied by profuse nasal discharge, at intervals of two or

three months. Patient came under observation January 7, 1903, suffering from dyspnœa and cyanosis, the attack having been precipitated by prolonged hard work in cold, damp weather, the patient drinking heavily during the time. Examination of the nose revealed polypoid degeneration of the ethmoid region in both nasal chambers, superior part of chloasmæ being filled with polyp tissue. The urine was 450 cubic centimetres, acid, sp. gr. 1.030, urea, 13.5 grammes, no albumen; the microscope revealed hyaline and granular casts. Physical examination of the chest showed emphysema of both lungs with sibilant and sonorous breathing and a few moist rales. The heart was enlarged, the first sound weak and the pulmonic closure accentuated. The pulse was increased in tension, of moderate frequency and size, and regular. The vessel walls in all palpable vessels were distinctly thickened. In this case, of course, the polypoid growths and the renal disease have to be considered, as well as the arterial degeneration, in their relation to the asthma and emphysema.

Another more typical case of asthma of arterio-sclerotic origin is the following.

D. M., male; physician; age thirty-one years; American. Father died of disease of heart and arteries. Mother living. Of nervous temperament. Personal history good, except for perityphlitic abscess discharge through bowel twenty years ago. In 1890, after severe struggle in a tug-of-war, he was seized with shortness of breath. Since that time he has had several attacks of wheezing and dyspnœa, especially after exhausting work. On January 15th he was seized with a distinct asthmatic attack. Physical examination revealed a mild degree of emphysema, asthmatic breathing, rales, heart slightly enlarged and, in spite of the asthma, the aortic closure accentuated. Urine 800 cubic centimetres, acid, sp. gr. 1.030, urea, 24 grammes, indican in excess; albumen, a trace; microscope, urates. Pulse full, tense, slow, regular. Vessel wall distinctly thickened. Attack relieved by sweats, nitroglycerine and potassium iodid.

Chronic fibroid pneumonia is not infrequently associated with arterio-sclerosis and may be considered a pulmonary expression of the disease in certain cases. Many of the pulmonary symptoms, however, such as dyspnœa, asthmatic breathing, bronchitis and œdema, are frequently referable to the morbid condition of the heart and kidneys rather than to that of the arteries themselves. Nevertheless, a sufficient number of cases remain, unaccounted for by kidney lesions, to give them a place in the semeiology of the disease. The symptoms of arterio-sclerosis referable to kidney lesions are those of interstitial nephritis of such grade as to interfere with proper excretion, varying from mild disturbance of cutaneous, digestive and nervous systems to those of pronounced uræmic poisoning.

The cerebral symptoms vary from mild degree of dizziness and lack of memory, particularly of recent occurrences, to violent vertigo, complete loss of memory and actual dementia, and

apoplexy; the eye symptoms are chiefly loss of vision, more or less pronounced according to the degree of involvement of the vessels of the retina. The group of symptoms included in the class that I have termed the cardio-vascular-hæmic are naturally the most interesting of the several groups, and the ones upon which the diagnosis ultimately must depend. The symptoms that I have mentioned as occurring in the several groups referred to may be dependent upon several morbid conditions and it is only when they are associated with those of the circulatory apparatus, and are relieved by measures directed to improving the state of the vascular system that we can consider them as symptoms of arterio-sclerosis.

In all cases of arterial disease beginning in the capillaries and arterioles—and almost all cases of arterio-sclerosis begin in that area—there is of necessity an alteration in the blood itself, partly due to lack of proper supply of nutriment and partly due to improper oxygenation and excretion, and partly due to the poison of the primary cause of the disease. Thus there is an anæmia of greater or less degree according to the primary etiological factor, the anatomical system that is most pronouncedly involved and the degree to which the arterio-sclerosis has progressed. The symptoms may vary from a persistently increased arterial tension with slight cardiac hypertrophy and increased intensity of the second cardiac sound as heard over the aortic orifice to the beaded or pipe-stem arteries, the greatly dilated left ventricle, the attacks of angina pectoris, marked dyspnœa, venous congestions and dropsies of the several organs and regions of the body, the evidences of pronounced cardio-vascular incompetence; or the complete occlusion of the lumen of a given vessel with fibroid or fatty degeneration, or gangrene of the part thus deprived of nutriment. It is my privilege, however, to call your attention to some of the earlier symptoms and signs of this widespread degenerative process. These symptoms and signs are especially associated with the capillary degeneration that causes rise in tension in the pulse. Therefore our first attention should be devoted to the recognition of this condition. While the recognition of increase in tension is of importance the other qualities of the pulse should not be neglected; for fully as much is to be learned from the careful study of the volume, frequency and rhythm as from that of the tension.

In almost all cases, except to some of those due to strain of occupation, the disease begins in the capillaries and consequently early in the disease we have a greater or less degree of cardiac hypertrophy. This hypertrophy of heart, together with the peripheral resistance, increases the tension and volume, slows the

rate and modifies the rhythm in such way that the vessel fills slowly, retains its volume for a longer than normal period and slowly empties itself. This character of pulse is an important early evidence of the disease and can be readily recognized if one uses three fingers in feeling the pulse, or follows Sansom's suggestion of laying one finger lengthwise along the radial pulse, supporting the wrist of the patient with the other hand.

The evidences of peripheral resistance to be noted in the pulse investigated in this way are as follows: The character of the pulse is to be noted with slight pressure of the fingers; if by increasing the pressure the pulse becomes more evident, and then by decidedly greater pressure on the part of the finger toward the heart it is imperceptible by the fingers beyond, it is a pulse of moderately increased tension; if, however, it is almost impossible or entirely impossible to shut it off by much pressure it is a pulse of greatly increased tension. Again the condition of the wall of the artery can be investigated at the same time. If we shut off the pulse by pressure with the finger nearest the heart, in a normal condition the artery collapses beyond and cannot be felt by the fingers toward the periphery; if the walls are thickened by the fibrous change, the vessel can be distinctly felt as a cord beyond. The sphygmograph, arteriometer and pulse pressure gauge in their present state of development are, in the opinion of the writer, not to be compared with the educated fingers in determining the character of the vessel or the quality of the pulse. At the same time with the increase in tension of the pulse the cardiac hypertrophy is usually demonstrable as is also the accentuation of the closure of the aortic valves. Both of these signs may, however, be interfered with if there is any considerable degree of pulmonary emphysema; and this, in the experience of the writer, is not at all uncommon.

Another relatively early symptom of arterio-sclerosis is the occurrence of vague pains or abnormal sensations, such as tingling, prickling, numbness or persistent coldness in various parts of the body, especially the limbs. These are frequently considered muscular rheumatism and treated as such. The appearance and persistence of such pains and the occurrence of sharp neuralgic attacks should call for the careful study of the pulse and blood vessels. These pains are due to malnutrition, the result of lessened blood supply through diminution of calibre or obliteration of capillaries, and the anæmia that I have already referred to as present in all these cases.

This malnutrition presents itself in two types, described by Mott as "the flabby and corpulent, and the sallow, emaciated, rather cachectic patient with *arcus senilis*. These patients fre-

quently complain of dyspepsia, flatulence, and intestinal troubles of various kinds."

In addition to these anæmic pains and to the characteristics of pulse and cardiac signs referred to, other symptoms referable to the anæmia and cardio-vascular apparatus are progressive weakness, shortness of breath upon exertion, præcordial distress or pain, vertigo and nausea. The following case illustrates this point well.

V. O.; male, twenty-nine years; German; married; laborer at a blast furnace doing heavy shoveling, lifting, carrying and hoisting. Family history good, except that father dropped dead; cause unknown. Personal history: habits, smokes occasionally, chews to excess; takes three to five drinks of beer or whiskey daily, but has never been intoxicated. No diseases until the present. In December, 1902, he stopped work because he felt tired and the work exhausted him. On January 7th, while walking was seized with dizziness, nausea and a sensation in the chest which he described as a "squeezing of the heart." These attacks recurred several times but were always relieved by lying down. Physical examination revealed a man of good build and muscular development, with a mild degree of anæmia (4,000,000 erythrocytes, seventy per cent. hæmoglobin). He was passing 1,100 cubic centimetres of urine, specific gravity 1.029, urea 33 grammes; no abnormal constituent revealed by microscope or chemistry. The walls of all palpable vessels were thickened though the tension was only moderate, the vessel tolerably well filled, regular, rate of eighty per minute. The heart was enlarged and slightly dilated; the first sound was weak and the aortic closure slightly accentuated. Although this case has not advanced far enough to show renal changes, the heart has already begun to dilate because the patient continued to work until he could do so no longer. It is an interesting case in that it shows that we may have serious cardiac failure even early in the disease.

I have thus briefly reviewed the symptoms of arterio-sclerosis as exhibited in the several organs, and systems of the body. The detailed description of each class is left to the succeeding speakers. In conclusion I would state, that if a patient presenting any one or more of the groups of symptoms described, shows in his history a hereditary predisposition to arterial disease, or has been exposed to one or more of the chronic intoxications, alcohol, lead, syphilis, gout and the so-called lithæmia, or shows evidence of renal disease or that his occupation exposes him to prolonged or severe muscular exertion, and the careful study of his heart, arteries and blood shows anæmia, abnormal sensation in the extremities, a pulse of increased tension, slowly rising wave with prolonged summit and slow descent, and slowness of rate with hypertrophy of heart, showing increase in length and strength of first sound and accentuation of the aortic closure, we are justified in our diagnosis of arterio-sclerosis.

III. SYMPOSIUM ON ARTERIO-SCLEROSIS

2. Arterio-Sclerosis and the Heart

By GLENTWORTH R. BUTLER, M. D.

BROOKLYN

The cardiac consequences of arterio-sclerosis may be divided into two groups, depending upon whether or not the coronary arteries are involved in the sclerotic process.

I. *Coronary arteries not involved:* If the coronaries are freely pervious and the heart muscle is in consequence well nourished, the main, and for a time the only, cardiac sequel of the general arterial disease consists in the development of a simple hypertrophy of the left ventricle. There may be little if any accompanying dilatation of the cavity. The obvious cause of the hypertrophy is the increased resistance offered by the thickened walls and narrowed lumen of the peripheral arteries whereby the work demanded of the left ventricle is largely augmented.

Cardiac hypertrophy is not a necessary result of arterio-sclerosis, even if the latter is diffuse. In the so-called senile form of the disease occurring in persons over sixty years of age, with thin-walled, large, and tortuous arteries, often rigid and of the "goose-neck" feel, the heart is frequently not at all enlarged, or the arterial tension increased. The subjects are usually of the feeble "old man" type.

Arterio-sclerotic hypertrophy reaches its highest degree in middle-aged, sometimes quite young, robust and muscular men. In this class the heart averages fourteen ounces in weight, and may reach twenty-five ounces. The subjects are the victims of some chronic intoxication, or are large eaters, or have been forced to do hard and prolonged muscular work.

In course of time the simple hypertrophy is succeeded by dilatation, accompanied by a train of symptoms which are almost exactly those of the broken compensation of valvular disease. The resemblance is so close, even to the presence of a loud systolic murmur at the apex, that the case, unless previously seen, readily may be mistaken for one of organic mitral insufficiency. If the heart is capable of at least temporary recovery by rest and other treatment the murmur will, by disappearing, reveal itself as a relative mitral insufficiency due to dilatation of the mitral ring with resulting inability of the valve cusps properly to coapt.

II. *Coronary arteries involved:* When the coronary arteries share in the general sclerotic process there may follow, with or without antecedent hypertrophy, fibroid degeneration of the heart, angina pectoris, coronary thrombosis with sudden death, and, rarely, aneurism or rupture of the heart. Thrombosis, aneurism and rupture are mainly of academic interest and will not here be considered.

Of these sequelæ chronic myocarditis is by far the most common. Indeed the fibroid heart practically is to be regarded as one of the diverse manifestations of general arterio-sclerosis, although it may arise from interference with the coronary circulation due to valvular defects, or may be part and parcel of a chronic endocarditis or pericarditis. The earliest symptoms which, when present, should arouse a justifiable suspicion of a progressing myocarditis are a sallow, pallid face, with enlarged venous radicles, premature grayness, a tendency to a wet and leaky skin, and a distinct loss of strength and energy. Moreover the pulse is apt to be weak by comparison with the strength of the apex-beat, and is irregular or intermitting. Frequently there are cardiac contractions of such slight strength that while they may be perceived by auscultation they are not represented at the wrist by a pulse-beat—ineffectual systoles. Frequent gastralgic attacks, presumably referable to the closely allied innervation of the stomach and heart, are to be regarded askant. In more advanced cases anginal attacks, with an irregular, weak, often slow pulse (fifty to thirty) are rather characteristic. When the fibrotic heart muscle is losing strength and dilatation is increasing, dyspnœa, præcordial constriction, palpitation, cardiac asthma, and the signs of general venous congestion become manifest. In some instances vertigo, syncopal attacks, and recurring, occasionally fatal, pseudo-apoplectic seizures distinguish the history of the disease. The physical signs in an advanced case are those of a dilated heart, with a galloping rhythm, often also the systolic apex murmur of relative mitral incompetency to which reference has previously been made.

True angina pectoris is a rather rare symptom. It is almost invariably dependent upon sclerotic changes in the coronary arteries and myocardium. The arterio-sclerosis may be purely local, affecting only the root of the aorta and the coronaries, or may be part of a wide-spread process. In deciding whether a given case of breast-pang is or is not a true angina pectoris an extreme importance attaches to the careful examination of the heart and vessels. Presumptive evidence of true angina may be derived from the age of the subject, the symptoms, and the mode of onset of the attack, but unless the physical signs of

sclerosis of the root of the aorta are present the diagnosis scarcely can be made. In cases of anginal pain attended by distinct thickening of the accessible arteries, increased tension, cardiac hypertrophy, and an accentuated ringing aortic second sound there is no difficulty in deciding that the attack is a true stenocardia. But when, as may happen, there is no cardiac hypertrophy and no palpable hardening, the judgment hangs entirely on the presence of heightened tension and especially upon the character of the aortic second sound. If the latter is accentuated, and if it possesses a peculiar, slight harsh or clicking quality at the instant of closure, a sign to be appreciated only by those reasonably well versed in auscultation, it is safe to decide that the angina is genuine and not false.

The gastralgic attacks to which reference has been made, have with me assumed an extreme importance lest they should be, at least the precursors, possibly the initial symptoms, of a true angina pectoris. The first case which led to suspicions occurred a number of years ago in the person of an elderly woman who was fairly embarked on the down grade of degenerative changes. She was under observation for some four or five years, and complained almost constantly of dyspeptic symptoms, such as slight nausea, eructations, and a feeling of epigastric distress. From time to time she would have, as the result of worry, fatigue, or dietetic indiscretions, model attacks of gastralgia without anginose symptoms. The latter were of course carefully looked for. Finally she had a typical seizure of angina pectoris with pain and tingling in the left arm and hand, restrained breathing, immobility, cold sweat, and intense fear of impending death. The latter premonition was unfortunately too faithfully realized by her sudden death in a second paroxysm two or three days later. Since then I have regarded with misgiving all cases of frequent marked or painful gastric attacks occurring in elderly persons who present evidences of myocardial and arterial degeneration. Four or five cases have served to confirm this impression. There is now under observation a man sixty-odd years old, of large frame and flesh, a tremendous eater, although not a consumer of wine or spirits, who has had within three years seven severe gastralgic attacks. His heart is moderately hypertrophied, his arteries noticeably hardened, and the aortic closure has a harsh clicking quality, significant of atheroma involving the aortic segments and perhaps also the coronary arteries. Of late this patient becomes dyspnœic upon exertion, accompanied by a marked sense of constriction in the chest, and his blood pressure is gradually rising. Moreover, at his last gastralgic seizure, a few weeks since, the pain radiated over the precordium and down

the left arm to the elbow, presenting for the first time a distinctly anginal character. According to all indications the case is tending directly toward a genuine angina pectoris.

Several instances of the curious and interesting condition known as the Stokes-Adams syndrome have come under personal observation. The pulse is permanently slow (twenty to forty per minute), and the slow pulse is associated with peculiar vertiginous, syncopal, apoplectiform, or epileptiform attacks. As a rule the patients are elderly, and are the subjects of far-advanced arterio-sclerosis. Among the several hypotheses put forward regarding the cause of this syndrome that which refers it to disease of the arteries of the medulla seems the most available.

Of the personally observed cases the most striking was one which occurred in the person of a man, sixty-eight years of age, a four-years' veteran of the Civil War. I saw him first at a time when he was confined to his bed because of dyspnœa and præcardiac oppression. He was propped up by pillows in a partly recumbent position, but sat upright and replied with much animation and many gesticulations to some preliminary inquiries. Without warning he suddenly fell back upon the pillows, the face became flushed, the eye-balls rolled to the left side, the pupils were contracted, and the respiration became rapid and stertorous, the cheeks puffing out with each expiration. In a moment the breathing gradually ceased, the face assumed a deadly pallor, the jaw dropped, the pupils became widely dilated and the pulse ceased. Neither by ear nor touch could any evidence of cardiac action be perceived. To all appearance the man was dead. Artificial respiration was immediately begun, but soon suspended while a stimulating hypodermic injection was prepared and given. After a lapse of time which is difficult to estimate, three minutes perhaps, a single pulse-beat was felt, followed by a feeble inspiratory effort, with a subsequent gradual increase in frequency and depth of breathing. The pulse-rate was, at first, four to the minute, then ten, then rose to sixteen within twenty minutes, and finally to twenty-four, at which it stayed thereafter. Consciousness gradually returned, being fully restored in about one hour, and there was no hemiplegia or other form of paralysis. Death occurred in a similar paroxysm about two weeks after the first seizure. During a part of this time the patient, a headstrong old man, occasionally rose from his bed and walked about the room. Examination showed him to be the subject of advanced arterio-sclerosis and myocardial disease.

IV. SYMPOSIUM ON ARTERIO-SCLEROSIS

3. Arterio-Sclerosis and the Kidney

By IRVING PHILLIPS LYON, M. D.
BUFFALO

General arterio-sclerosis and chronic interstitial nephritis frequently occur together in the same subject. Either may be the primary affection and produce the other as a secondary process. On the one hand, chronic interstitial nephritis, as a primary disease, leads to increased arterial tension, which in turn by its strain causes sclerosis of the arteries. On the other hand, general diffuse arterio-sclerosis, as a primary affection, may ultimately invade the kidney and produce its chief effects on this organ, causing either a patchy or a diffuse sclerosis, *i. e.*, chronic interstitial nephritis. Whichever disease is the primary one, the ultimate effects may be similar or identical.

The problem for the physician is to determine, in a given case, the primary from the secondary process, the cause from its effect. What differential criteria for this purpose are available? The chief points that may assist in the determination are found in the history of the case, the degree of arterio-sclerosis compared with the amount of the renal impairment, the sex, the age, and the course of the case while under prolonged observation.

In the history, all general and specific causes of arterio-sclerosis should be sought and weighed with reference to the sex, age, degree of arterio-sclerosis and urinary changes. Among such causes, syphilis, alcoholism, plumbism, gout and long-sustained, severe physical strain are of chief importance. These are the most common causes of marked arterial degeneration and, as such, point strongly to the arteries as the probable primary seat of disease. A marked history of these factors, one or more, may be decisive in the diagnosis, when considered with reference to the case in hand. Syphilis and alcoholism are the most frequent causes of marked arterio-sclerosis and can hardly be overestimated in importance.

Sex is considered simply because the male sex is predominantly exposed to the action of the above-mentioned chief causes of arterial disease. Exceptionally any of these causes may be active in females and in such cases the history is usually positive.

The degree of the arterio-sclerosis compared with the amount of the renal impairment and the known course of the latter is the chief criterion. The history helps in this connection by furnishing a certain and plausible explanation of the arterial thickening. If the arteries show high-grade thickening, out of due proportion

to the apparent kidney lesion, the case is one, probably, of primary arterio-sclerosis.

The course of the case under prolonged observation may materially strengthen such a judgment. If, when the patient is first seen, the arteries are hard, perhaps tortuous and nodular, and the urine shows slight or only transient pathological changes and under observation for months or years there is very slow and slight increase in the renal lesion, such evidence from continued observation confirms the opinion formed on the first examination.

The age of the patient throws further light on the problem, for he is likely to be a young or middle-aged adult, if the arterial disease is a primary factor. At least this is often the case in those instances in which a differentiation of the primary from the secondary seat of disease is still practicable. This is explained by the fact that syphilitic and alcoholic arterio-sclerosis is developed usually in early or middle life, before the event of senile degeneration. If, on the other hand, a less marked and more slowly developing arteritis extends into advanced life the distinction between primary and secondary arterial disease may be difficult or impossible.

We may conclude, then, that a case is one probably of primary arterio-sclerosis and secondary nephritis, when, with a history of syphilis, alcoholism, gout, plumbism, etc., in a young or middle-aged adult, usually a male, we find an advanced stage of arterial thickening out of due proportion to the apparent degree of renal impairment, especially if the latter shows only slight and slow increase during prolonged observation.

What practical value has the differentiation which we are attempting to make? None, so far as I am aware, in advanced cases in which treatment can be of little value, more, certainly, in less pronounced cases, particularly in men before the turn of life. In such cases we would not alarm the patient with a diagnosis of Bright's disease, nor would we treat the case as one of nephritis. We would not add to the strain of the stiffened arteries by ordering kidney flushings, sweat-baths, and similar drastic measures. We would not mistake a pulse of small volume for one of low tension and prescribe digitalis, as is so frequently done in these cases. We would not annoy the patient by too severely restricting his diet. We would frankly warn him of his true condition, that his arteries are older than his body by many years, that he must lead a quiet, rational, well-regulated life, avoid unnecessary excitement, strain, worry and fatigue, protect himself from wet and cold and sudden changes of temperature, dress warmly, eat and drink in strict moderation, give up all stimulants, protect his arteries from all undue strain, and that,

if he will observe all these precautions, his chances of a reasonably long life are measurably increased. And finally we might use with benefit in selected cases for long periods of time such drugs as nitroglycerine to reduce high arterial tension. Such, then, are the practical features of value in correctly interpreting the primary seat of disease, which should warrant the more general recognition by practitioners of the distinctions indicated.

V. SYMPOSIUM ON ARTERIO-SCLEROSIS

4. Arterio-Sclerosis and the Digestive System

By CHARLES G. STOCKTON, M. D.
BUFFALO

The subject of arterio-sclerosis, in its causative relations to diseases of the digestive apparatus may be considered under two heads: first, when, as a direct result of the sclerosis of vessels, arteritis obliterans or endarteritis, there follows a definite lesion of one of the digestive organs; second, when arterio-sclerosis, having produced disturbances in the heart, lungs, brain or kidneys, there are set up, as secondary manifestations, either structural or functional diseases of one or more of the organs of the digestive apparatus.

FIRST—LESIONS RESULTING DIRECTLY FROM ARTERIO-SCLEROSIS.

Aneurism of the descending or abdominal aorta or of its branches may exercise pressure upon the esophagus, the stomach or intestines, and thus give rise to obstruction; from this may ensue simple disturbance in function, or the pressure may give rise to atrophy, ulceration and loss of substance, accompanied by a train of painful and serious symptoms. Especially during the functional activity of the esophagus, stomach or intestine are these symptoms prominent. Not only does digestion become painful, but through dread of suffering, there is voluntary abstinence from food on the part of the patient.

Embolism or thrombosis of the vessels supplying the abdominal organs may give rise to serious results. This is particularly true of the classic obstruction of one of the mesenteric vessels when there develops gangrene of the intestine and a chain of symptoms not always correctly interpreted. The condition has been mistaken for acute pancreatitis, obstruction of the bowels, appendicitis, perforation of the intestines, etc. The real diagnosis is often overlooked. One usually finds abdominal pain and tenderness, vomiting, moderate tympany, tension of the ab-

dominal walls, and diarrhœa accompanied by marked constitutional symptoms, briefly described in the phrase "abdominal shock."

Gastric Ulcer, it has been held from time to time, may depend upon arterio-sclerosis. Virchow long ago called attention to the fact that thrombosis of vessels in the gastric mucosa, leading to hemorrhagic necrosis, would, in the presence of active gastric juice, result in a peptic ulcer of the stomach. This he demonstrated, and it has subsequently been shown at numerous autopsies. Of course, this accounts for only a small proportion of all gastric ulcers. Arterio-sclerosis, however, is by some held responsible for another form of gastric ulcer, and the subject is undergoing discussion in recent literature. I refer to a condition in which there is practically obliteration of a comparatively large branch of one of the vessels of the stomach so that inosculation is made impossible and a relatively large tract of the gastric mucosa is deprived of arterial blood. This condition must be rare for its occurrence is denied by some experienced clinicians. Riegel, for instance, refers to it, but doubts its existence. This was my own view until I saw the evidence presented by Dr. H. U. Williams at a recent meeting of the Buffalo Academy of Medicine. The patient, a woman, forty-eight years old, died at the Erie County Hospital of what was presumed to be carcinoma of the stomach. At autopsy marked arterio-sclerosis was found, aneurism of the arch of the aorta, and, as a result of the obliterating arteritis, the blood supply to a considerable area of the gastric mucosa was interrupted. In the center of this area was a large gastric ulcer from five to eight centimeters in diameter, irregular in form, but having the characteristic punched-out appearance, without evidence of inflammatory reaction at its border. This is one of the largest ulcers that I have ever seen. From careful examination of the specimen I am convinced that it resulted from the cause above described, and it may be possible that the very large ulcers of the stomach, occasionally observed past middle life, result from this condition of arterial disease more often than is generally supposed.

In the *Wiener klinische Wochenschrift* for September 18, 1902, Neusser of Vienna has reviewed the gastro-intestinal disturbances that occur in arterio-sclerosis and has collected some of the literature on the subject. Schrötter is quoted as describing a number of cases in which *meteorism, colic and other disturbances* were apparently traced to deficient blood supply in the regions involved. These attacks developed when the functional activity of the part was put to strain. He likens the behavior of the intestine to that of angina pectoris when the myocardium is in a

state of ischemia from coronary disease. He found that his patients improved by the administration of the nitrites. The conclusions reached by Schrötter seem to be logical, and since reading his article I have had personal experience with a case which I believe is somewhat similar. The patient, a middle-aged woman, complained of pain in the region of the umbilicus two hours after eating. There were also some meteorism and a sense of stoppage. Her gastric secretion was somewhat depressed, but this did not seem to me adequate to account for her symptoms. She was not relieved by dieting nor by the usual remedies employed for her gastric condition. She was, however, promptly benefited by nitroglycerine. In the same article Ortner is quoted as having observed a similar case in which it was particularly noted that there occurred lessened peristalsis. At autopsy there was found atheroma of the ascending aorta, abdominal aorta, arteries of the stomach, and also of the superior mesenteric artery. The subject is so new, and the evidence afforded by autopsy is still so meagre that it is unwise to come to an immediate conclusion concerning it. It is not improbable that many cases of abdominal disturbance may depend upon the direct results of arterio-sclerosis; but it must not be forgotten that in these same cases, the indirect effect of circulatory disease may produce symptoms quite similar in character. When, however, it is found *post mortem,* that the intestinal blood supply was greatly diminished as the result of vascular disease, it is proper to assume that the history of abdominal symptoms is to be explained by arterio-sclerosis.

Cases of marked abdominal symptoms in which it is doubtful whether these arise from the direct or indirect result of arterio-sclerosis. There is a comparatively large group of cases of arterio-sclerosis that suffer from symptoms referable to the stomach or the intestine, or both, and which from one point of view seem to depend upon insufficient blood supply to the digestive organs. But from the fact that at the same time these cases suffer from general circulatory disturbance as well as from renal insufficiency, it seems hardly warrantable to assume that the symptoms are solely the result of local ischemia in the digestive tract. This subject is best illustrated by the report of cases.

Case I. An aged physician who saw service in the Civil War, where he suffered from malaria, was subsequently in perfect health; a splendid specimen of manhood, six feet tall, weighs 206 pounds, and in most respects shows good preservation. For two or three years past he has been somewhat short-winded in walking rapidly or going upstairs. For several months past he has slight dyspnœa and occasionally precordial pain caused from excitement. One month ago, after a hearty evening meal, he went

to sleep in an open camp. The night became cold and he was awakened with intense pain in the precordial region, dyspnœa and vomiting. He had previously had attacks of pain which had been relieved by vomiting; this time the vomiting did not relieve him. He took whiskey and succeeded in reaching home in a very depressed condition. During the succeeding four weeks, until he visited me, he abstained from eating solid food and took liquid food scantily. He was especially afraid of starchy food, and felt positive that solids and the starches in particular caused pain in the epigastrium, accompanied by shortness of breath. The physical signs showed arterio-sclerosis, the left border of the heart just outside the nipple line, the apex somewhat diffused, with an unusual cardiac impulse in proportion to the cardiac sounds, which were feeble. The first sound at the apex was especially weak, the second louder and of a very high-pitched, ringing character. The sounds at the base of the heart were feeble, the aortic second was extremely high-pitched and valvular. The heart was intermittent and irregular in action; the area of hepatic dulness was somewhat narrow. The urine was not diminished in quantity, had a specific gravity of 1.011, but contained only twelve grammes of urea in twenty-four hours and an occasional hyalin cast. He was treated by frequent minute doses of nitroglycerine, 1-400 of a grain hourly, and the application of thermal baths. Subsequently, small doses of potassium iodide were given. The diet was progressively increased, and after three weeks he returned home able to eat a moderate meal in comfort. He has since informed me that he is able to eat provided he does not exercise, and to exercise provided he does not eat.

The second case illustrates a somewhat different phase.

Case II. E. C. W.; aged forty-six years; manufacturer; denies venereal disease. He first consulted me in 1896, complaining that for the past year or more he had had attacks of abdominal pain and distension coming on about midnight, accompanied by restlessness. His physician had to be summoned to relieve him by hypodermic injections of morphine. He showed a very marked arterio-sclerosis; had lost in weight and color. The pulse, though tense, was frequent; the cardiac apex diffused, extending one-half inch to the left of the nipple line; the aortic second sound was *very* much exaggerated; there was no bruit. He voided sixty-four ounces of urine in twenty-four hours, containing sixteen grammes of urea, and an occasional hyalin cast. The stomach contents, three hours after a test meal, showed a total acidity of 111, of which 71 (or twenty-five hundredths per cent.) depended on free hydrochloric acid, combined chlorides 20, and acid salts 20. Alkalies were given for the hyperchlorhydria, potassium iodide in small doses, and thermal baths for the relief of the arterio-sclerosis. It is now seven years since the patient first consulted me. He has been free from trouble so long as he has occasionally resorted to the original line of treatment; sometimes it has been found necessary to give him nitroglycerine. It is fair to state that his symptoms were not those usually seen in hyperchlorhydria. I am not satisfied that the renal inadequacy was sufficient to produce these atacks. I am inclined to think that the arterio-sclerosis has given rise to intestinal ischæmia

and is chiefly responsible for his seizures. It should be noted that the complaint was mostly intestinal distension and pain below the stomach, the bowels at the same time becoming inactive. Such cases, while not common, are met with sufficiently often to make further reports unnecessary.

SECOND—GASTRO-INTESTINAL DISORDERS INDIRECTLY RESULTING FROM ARTERIO-SCLEROSIS.

Arterio-sclerosis is indirectly a common cause of gastro-intestinal disorders. To prove this it is necessary only to cite the familiar condition of *secondary dilatation of the heart, and succeeding venous stasis,* which gives rise to distension of the portal system and congestion of the abdominal viscera. Irritation and mild catarrhal inflammations of the stomach and intestine are met with, and disordered secretion and disturbed motion are commonly present.

Another group of cases is to be attributed to the *interstitial nephritis* which so often forms a part of general arterio-sclerosis. The resulting uremia gives rise to a great variety of symptoms both in the stomach and intestine, which are so well recognized that it seems fruitless to review them here.

There is *a form of colitis* to which Delafield some years ago directed attention, which appears to be the direct result of chronic interstitial nephritis, or in other words, of arterio-sclerosis. The colitis is of an ulcerative type, and not infrequently proves fatal, but clinically is represented by the familiar picture of chronic colitis. I have known cases in which the intestinal symptoms were inconspicuous, although the lesions of the colon were unquestionably the direct cause of death, as shown *post mortem.*

Coronary disease of the heart and the digestive apparatus. Among the direct effects upon the digestive apparatus produced by arterio-sclerosis is the somewhat unusual one of intense abdominal pain generally referred to the pit of the stomach and closely resembling gastralgia. The first case which came under my observation was seen in consultation with Dr. DeLancey Rochester of Buffalo, and was that of a woman about sixty years old who was seized in the night with terrific gastralgia which was not easily controlled. The heart action was not especially disturbed, and this organ at first was not suspected as the seat of trouble. The pain recurred from time to time during a period of about three days when the patient suddenly expired. The post mortem revealed an extensive coronary disease with rupture of the left ventricle near the apex, and the over distension of the pericardium with blood escaping through the ruptured ventricular wall. A second case, almost precisely identical, I had occasion to observe in the person of a well-known physician of western New York. The particulars of two other very similar

cases have come to my knowledge. In the first case there was nothing to attract attention to the heart, but in the second case the proper diagnosis was made. The only gastric symptoms present were those of gastralgia.

CONCLUSION: In concluding this brief paper, I wish to say that I have gone over with considerable care the recent cases in my record book to find the proportion of cases of arterio-sclerosis in which complaint was made of symptoms of disease of the digestive apparatus, and find that the proportion is large, namely about sixty per cent. This proportion, however, is misleading for the reason that many of these patients consulted me primarily because of symptoms of abdominal disease. It would be interesting to know the frequency with which gastro-intestinal disorders develop in all cases of arterial disease. The belief is growing that arterio-sclerosis is more frequently the direct cause of disease of the stomach than we had formerly supposed. As an indirect cause of many digestive disorders the arteries have long been held justly responsible.

VI. SYMPOSIUM ON ARTERIO-SCLEROSIS

5. ARTERIO-SCLEROSIS AND THE NERVOUS SYSTEM

BY WILLIAM BROWNING, M. D.

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OF recent years neurologists have been giving an increasing share of attention to this condition and its results. In fact, as so often with subjects, even scientific, that are being boomed, a distinct inclination may be noted to do as the politicians, "claim everything" under this head. A portion of these sweeping claims may prove warranted, but it is certainly time for conservative judgment.

If we exclude from consideration the grosser secondary effects (as softening, hemorrhage, etc.), is it possible to isolate a picture of arterio-sclerosis pure and simple, to specify manifestations directly attributable to that condition or its immediate effects? This might be possible, even though the disorder never, in fact, occurred in a pure form. There are, however, often doubts whether these manifestations are of such simple mechanical origin, especially as certain agents not only cause this trouble but are also capable by themselves of producing nervous disturbances. It must consequently be granted that an element of uncertainty still exists with regard to many of the interpretations, even to such as are based on pathological findings.

If, instead of saying that these symptoms are due to arterio-sclerosis, we tentatively say merely that they are more or less often associated with it, we can proceed with the subject unhampered.

The most that can be attempted here, besides offering a convenient classification, is to summarize the symptoms attributed to this cause and briefly comment thereon. It will not be possible to consider different stages nor finer subdivisions that have in part been proposed. The selection of type forms does not, of course, preclude the occurrence of intermediary and irregular cases.

It is recognized that this process may expend itself specially on the cerebral vessels. And, more than this, that it does not equally affect all the vessels even there.

Doubtless clinically we as yet bunch a number of more or less distinct processes,—atheroma, arteritis and endarteritis, perivascular gliosis, arterio-sclerosis, etc.,—under the one heading.

1. In the first place, changes in the peripheral nerves have been described that were attributed to local disease, in part arterio-sclerosis, of the vessels of the extremities. The nerve trouble was interstitial rather than parenchymatous, however, and has little relation to the other forms to be described. (Lapinsky, *Deutsche Zeitschrift für Nervenheilkunde,* 1898.)

The condition called intermittent limping or claudication (angina cruris of Walton) is found to depend on local arterial changes, in part also of this character,—though calcareous arteries, nicotinism, rheumatism, etc., are more important factors.

As a whole, these peripheral matters have not as yet assumed much clinical importance.

2. Next, we come to the spinal symptoms of arterio-sclerotic origin. These in part have been attributed to involvement of the spinal vessels themselves, in part to that of vessels higher up.

Demange was, perhaps, the first to call attention to this phase of the question. He reported autopsies in some cases (*Revue de Medicin,* 1884, No. 10,—1885, Nos. 1 and 7). He figures diffuse scleroses not of the insular form as definitely of arterio-sclerotic origin. Clinically a symptom-picture analogous to that of lateral sclerosis was produced by the morbid process.

It was to cases of this kind that the writer referred when discussing the pressure effects of the column of cerebro-spinal fluid in the upright (page 20 of "Circulation in the Central Nervous System"). "In the feeble and senile the finer nutrition of the yielding structures of the lower cord and its roots may indeed suffer. This suggests itself as one cause for certain cases of senile paraplegia, in which the upper extremities are fairly in-

tact.'' Eventually, however, the upper extremities do show motor weakness and decided increase of the tendon reflexes.

Last year Hirsch, of New York, described something of this kind at the meeting of the American Neurological Association.

Without doubt these accounts refer to a definite clinical group of cases, whatever may be their source or explanation. The subjects are usually persons of fine physique, but in the declining period of life. The tendon reflexes of the lower extremities and then of the upper become decidedly exaggerated both in force and numbers, although without clonus. An increasing muscular weakness accompanies this, but no loss of sensation. For a time the mental functions appear unimpaired, but later in the case may suffer some also in a very general way. The gait is shuffling rather than pronouncedly spastic. As has been suggested, this condition might be due to interference higher up than the cord. Yet except for the late mental impairment, we see something not widely different from this type in spastic spinal syphilis. And in one of my cases the woman's father had died of tabes.

Therapeutically it is noticeable that these cases yield to a different line of remedies from those of most use in the brain-forms. Vaso-dilators, the strychnine group, strophanthus, are specially helpful and often serve to tide the subjects along for an added couple of years of activity or tolerable existence.

Other spinal forms of arterio-sclerosis including systematized cord-troubles (Redlich) have been described, but can not be dwelt upon.

3. We now come to the larger question, viz., that of the brain-forms. It is convenient here to distinguish a progressive or severe type, and a common or simple and better known form.

I. In the former the symptoms are gradually progressive or run on in a fairly even manner, and the cases take an earlier fatal ending. It is here also that we may class the organic paralytic forms. Here are many cases like those studied by L. Jacobson, in which the end-arteries to the pons or to the internal capsule are specially involved; in such it is but natural that striking permanent injury should occur.

From various histories it appears that this type often shows a syphilitic or traumatic etiology. Such cases are not much responsive to treatment, even if instituted before definite injury.

II. In the ordinary and best-known type the manifestations are of varying character and importance. One time with another a multitude of symptoms may be noted, some of minor account. Many of these may set in suddenly and within a few minutes, hours or days subside leaving nothing definite or only a minimum of the threatened trouble. These, however, either tend to recur,

or in some cases may be more or less continuous. Whatever is left from time to time depends presumably on the limited extent of permanent damage done before circulation was re-established in some area.

The signs suspected of having some relation to or to be suggestive of this condition include: dizziness usually slight and diminished by reclining, feelings of nausea, or rarely there may be actual vomiting, tinnitus aurium with changes in audition even to the hearing of distinct sounds, transient aphasias,[1] also paresis or paræthesias of an extremity or side, motor weakness of both sides (di-paresis), a tendency to pitch or fall, irregular clumsy or tremulous movements, faint turns, hebetude and sleepiness, especially dropping asleep in a chair or at work even in the forenoon, an inability to stand much loss of sleep, less often insomnia, flying pains about the head, particularly the forehead, or more general headache, a feeling of pressure or heaviness in the head, impairment of memory, "nervousness," rarely periods of irritability, great excitement or mental confusion, lack of accustomed energy, failure of quick mental initiative power, increased obstinacy, a tendency to depression and melancholia or great fear of losing the mind, intolerance of mental strain, alcohol or tobacco although there may be an increased craving for stimulants; in a few cases epilepsy or convulsive phenomena, not rarely Jacksonian in character ("senile arterio-sclerotic epilepsy"), sometimes an injurious influence from hot weather, etc. Until the condition, however, is advanced there is not usually any gross impairment of mentality. The particular symptoms in an individual case depend largely on the areas involved.

There may be ocular symptoms of great import. This is but natural, as the retinal supply comes from the cerebral carotid and is closely analogous to that of the brain itself. Some of the special anatomical conditions favoring this have recently been studied by Liebrecht (*Archiv für Augenheilkunde*). And Otto has described four cases depending on pressure by the changed carotid or ophthalmic artery on the optic nerve.

Flickering before the eyes or transient blurring of vision, and, according to Vogt (1902), contraction of the visual field in most of the advanced cases and sometimes in others presenting definite evidence of arterio-sclerosis.

Of great diagnostic importance have been found special alterations, haziness, etc., about the retinal vessels. There may be "A tortuosity of the *smaller retinal* vessels, and of their *terminal twigs.*" Perhaps allied to this is the occurrence of transient spasm in the retinal vessels (Dr. Ole Bull), or local and fleeting

1. The more highly organized and delicate the brain function the more likely it is to suffer involvement. Hence passing aphasias, agraphias and incoordinations are not uncommon.

contractions of the same. It has become a matter of prime importance to have the aid that can be furnished by a competent ophthalmologist, towards early diagnosis and the corroboration of our suspicions, particularly where imperfect elimination is in play (Raehlman, unpublished paper of Alleman of Brooklyn, and others).

As many of these manifestations are similar to those seen in neurasthenia the possible identity of the two, at least in certain cases, is claimed by some authorities, while other good writers argue strongly against this view or take a middle stand. It is at about the middle period of life that many cases of neurasthenia occur, and it is at about that time that arterio-sclerosis often begins to make itself felt,—although in many cases as now appears, beginning even much earlier. It must be granted that incipient arterio-sclerosis plays a part in many cases classed symptomatically under neurasthenia,—a fact not at all negatived by their apparent recovery.

Much the same statement applies to the so-called traumatic neuroses. The development of serious trouble in consequence of slight trauma has been claimed (*e. g.* by Windscheid, *Münchener medicinische Wochenschrift*, 1902) to depend upon previously existing sclerotic changes in the brain-arteries. This may apply to certain severe cases. But it is evident that in the traumatic-neurotic the disturbed vaso-motor innervation must lead to abnormal variations of arterial pressure and of itself favor the development of arterio-sclerosis.

The interpretation of the various symptoms enumerated depends largely on their relation to a number of co-ordinate matters quite as definitely suggestive of advancing arterio-sclerosis, as well as on the existence of known causes. A few may here be mentioned.

It will be seen that many of these signs might merely indicate senility or the presenium, or be due as above noted to the direct action of some of the causes of the arterio-sclerosis itself (uric acid, senile lithæmia, sluggish excretory functions, abuse of alcohol, nicotinism, plumbism, syphilis, sexual excesses, overeating, etc.). Old age favors it, although middle life and even earlier may not be exempt especially where heredity plays a rôle (E. D. Fisher, and others).

As to uric acid, it is just possible that the cart is sometimes put before the horse. Clinically it appears so often evident, not that uric acid has to any great extent caused the outbreak of symptoms, but that the nervous strain and overwork have developed uric acid, the latter in turn doubtless augmenting matters somewhat.

As to arcus senilis, I am forced to doubt its bad omenship. Tortuous temporals and hardened radials have but a limited bearing, for as stated by Alzheimer (*Neurologisches Centralblatt*, 1902, p. 420), "The parallelism between arterio-sclerosis of the central nervous system and of the remainder of the body is far from being constant."

When the process is far advanced and diffuse, a general increase of the tendon reflexes, particularly those of the lower extremities, is common.

Besides these points we at times find aid from the various circulatory, respiratory, cutaneous and allied matters considered by the previous speakers.

TREATMENT

1. The open-air life. This usually carries with it a certain amount of light exercise.
2. A let-up of mental and physical tension, but not always entire relaxation from activity.
3. Laxatives, particularly salines and sulphur waters,—the latter only for specified periods.
4. Proper hours of sleep.
5. Limitation or interdiction of alcohol and tobacco.
6. Some regulation of diet.
7. Anti-lithic remedies, including the well-known iodide.
8. In some cases certain drugs already mentioned.

We hear mostly of its bad portent. But it is well to remember that, while it is a degenerative or senile affair, it is not necessarily without great remissions in symptoms, especially when recognized and cared for, and may continue as merely a handicap for many a year. This fact is established not only by clinical but by pathological evidence.

VII. SYMPOSIUM ON ARTERIO-SCLEROSIS

6. Arterio-Sclerosis and Mental Disease

By ADOLF MEYER, M. D.

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Among the functions of the nervous system there is one large group of vital importance, but at the same time of perplexing difficulty of explanation, the mental life of the individual. That we should single it out from among the other functions of the nervous system, has its good reasons. It is partly because the mental life is in itself the most important function, and because it depends on a fairly definite part of the nervous system, one much more difficult to understand in its totality than the peripheral nerves and spinal cord or medulla and the so-called motor and sensory centers by themselves. This singling out comes moreover from the fact that the study of mental disorders is almost exclusively left in the hands of the few physicians who devote themselves to psychiatry. Physiology and medicine give little instruction to the practitioner in these directions, probably because they still are too complicated and not easily made teachable. The arrangement of this symposium left this most difficult topic to the last, and it did so with good justification. There is not much to be offered that would be of much value from the point of view of treatment of arterio-sclerosis. The *consequences* of the effects of arterio-sclerosis on the mental functions are, however, of great *practical* importance and we shall briefly review them.

We have heard of the rôle arterio-sclerosis plays in the life of the blood-vessels, the life of the heart, the life of the kidney, the digestive system, the nervous system in general. Like every other organ the part of the nervous system which is the organ of mental functions, has its distinct biotrophy or vital elasticity characteristic of the individual and often enough of many members of the same family. It ages more or less independently, and it may, moreover, suffer through arterio-sclerosis in chiefly the following three directions:

1. From the effects of a reduction or change of metabolism due to arterio-sclerotic disease of some other organ or organs—the heart, the kidneys, etc.;

2. From disease of its own vascular mechanisms, especially sclerosis of the larger or smaller vessels of the brain;

3. Or it may remain behind in its own metabolism owing to exhaustion of the vital elasticity of the nerve-tissues proper, especially when more trying demands are made upon it through toxic substances or stress. These factors should be considered in a diagnosis, if by diagnosis we understand not a mere name but a statement concerning the nature of the disease, the causes, and their working, and possible clues concerning means of repair.

Our first question is therefore: Are there any mental disorders due to arterio-sclerosis of other organs while the brain as such shows no arterio-sclerosis?

Such disorders occur, but they are usually symptomatic, *i. e.*, they disappear with the removal of the cause, and they are not very frequent. I refer to certain types of delirium known as uræmic delirium which, in rare cases, may last a considerable time. These states do not really differ from delirium in states of uræmia of non-arterio-sclerotic origin. I am, however, strongly under the impression that the frequency of mental derangement attributable to lesion of the *kidney*, is usually exaggerated. While, undeniably, the persons who show arterio-sclerotic involution of the brain usually also show the same disorder in the kidney, the reverse is hardly the rule.

Arterio-sclerosis of the heart and the aorta are also very frequent in the insane; but it is not often that we can lay any mental disorders directly to the door of these affections. Disorders which might belong here are difficult to explain; thus I have seen embolism from the aorta with apoplexy and hemiplegia and death preceded by several days of delirium, without there being any evidence of an embolism preceding the fatal one.

We only begin to have safe ground under our feet where we have reason to suspect arterio-sclerosis of the brain itself, and even there we meet with much difficulty when trying to distinguish between what is to be accounted for by the blood-vessels and what by the simple abiotrophy, or senile reduction, of the organ and its function, in which arterio-fibrosis is merely a concomitant fact.

We briefly review the forms of insanity occurring after the age of forty-five, and ask in which we have reasons to suspect cerebral arterio-sclerosis?

First we meet with all those forms which may occur at any age; alcoholic insanity, general paralysis, usually due to a previous syphilitic infection: further, exhaustion-dileria, and that large group of patients who have a number of attacks in their life usually depressive or exalted or mixed, *i. e.*, the cases of

manic-depressive insanity; further forms of paranoic or delusional states, and forms of deterioration.

As specially characteristic of that period we find anxious melancholia, and certain paranoic states of a hypochondriacal basis or turning around property questions. Of all these diseases we know that they may occur without any arterio-sclerosis.

The specifically senile psychosis practically always associated with arterio-sclerosis, is, however, one of general mental reduction and apparently a fairly independent condition. The senile reduction of the mind is a phenomenon from which few people escape completely in old age, and which is apt to show prematurely in some. This is usually part of the general senile involution. We possess very interesting studies on the effect of this process on the mental functions. In ordinary life we notice the decrease of memory, a tendency to follow ruts of thought and to rehearse them, a certain narrowing down in interests and in the grasp of new things. Ranschburg has made a careful psychological comparison between the functions in ten young men and eleven old men of the same level of education and society. He found that the old men perceive sensations about thirteen per cent. more slowly, reactions involving choice take forty-four per cent. longer than in the young; in simple calculations the old men were thirteen per cent. slower, but they made slightly fewer mistakes. The slowness may be accounted for by the greater slowness in reading; obviously, calculation does not suffer as much as the hearing and reading of the figures. Judgments tested by the question whether a name shown referred to something living or lifeless were given seventeen per cent. more slowly by the old, and the mistakes were twice as numerous as in the young. The drift of associations of words is fifty-four per cent. slower and the associations are made very largely according to sense and especially according to causal or purposive relations, not according to similarity of sounds or other free or accidental relations. There is obviously less elasticity and a limitation to relations which experience has impressed over and over again. This is in harmony with the reduction of free imagination in old age. This general factor which seems to be independent of actual arterio-sclerosis marks most old people.

Without evidence of a certain definite form of deterioration and dementia, the various forms of mental disorder have *relatively* the same prognosis as in earlier life, if at least we make allowance for the increasing loss of recoverability in old age. Well-founded suspicion of arterio-sclerosis exist, however, when an ominous deterioration begins, with a gradual loss of memory, a narrowing of judgment, and transitory states of confusion,

often with delirium of occupation, and without the cardinal symptoms of general paralysis.

The loss of memory which affects mostly the immediate past makes the patient repeat himself and leads to difficulty of grasp on the situation; the patient loses himself temporarily, gets confused, and especially at night, is unable to find himself, and is apt to develop outbreaks of anxiety, often with frightful hallucinations. At times the gaps of memory are made up for very liberally by confabulations. The patient does not remember what time of the day it is, but says he has just been down town, claims that friends have just been there, etc. Frequently he shows an aimless desire for activity, and in the confusion he rummages in the drawers or collects useless articles and conceals them in the clothes or beds; or one of my patients who was invited to dinner, failed to come to the parlor and it was found that he had undressed and gone to bed, instead of simply putting down his overcoat and getting ready for dinner. Or, even in the beginning of the disorder, criminal acts occur, such as exhibition of the sexual organs, or other sexual misdemeanors, stealing, putting fire to places through carelessness. These not infrequently are the outcome of the peculiar perplexity or bewilderment which shows also in the fact that the memory for the act may be missing, as in an otherwise fairly preserved case at the Worcester Hospital; a patient who killed his wife, and continually wondered why she did not come to see him. Not infrequently the delirious states are transitory at first, chiefly nocturnal, whereas the fundamental defect of memory and judgment which is not accounted for by temporary confusion and perplexity, is as a rule lasting or progressive.

We often see increasing self-absorption with hypochondriacal complaints partly built on the natural fear of becoming unable to control the sphincters, but also exaggerated to claims that the internal organs are absent, stools have not passed for months, etc. A feeling of helplessness leads to ideas of persecution, fear of being mutilated. Property questions are prominent and are apt to show the patient very unreasonable.

In the prognosis of mental disorders in old people the existence of arterio-sclerosis of the radial, of the kidneys, arcus senilis, tremor, senile atrophy of the skin, tottering gait, are not absolute evidence of similar involvement of the brain, but must be taken as evidence of a serious danger. Certainty of a process of cerebral atrophy comes only where we have positive signs of dementia or focal disease.

It is impossible to-day to say to what extent cerebral arterio-sclerosis as such is actually the *cause*, or simply a *part*, of the

general involution. After all, the only symptoms which can be laid more directly to the changes in the blood vessels are those of a focal nature, which have already been discussed by Dr. Browning, such as the occurrence of apoplectic attacks of various extent, the appearance of symptoms from focal lesions and the vaguer symptoms, such as circulatory difficulties, dizziness, and occasionally neuralgias of a whole side of the body. My conclusion concerning the insanity would be that we have no means at present to speak of arterio-sclerotic insanity, but only of insanity of senile or prematurely senile involution, and that the real arterio-sclerotic nature is only revealed by the course, and by the nervous and collateral symptoms of focalized or general arterio-sclerosis. The so-called organic dementia following apoplexy is usually a compound of the effects of direct focal destruction of brain and diffuse changes. In rare cases though, it would seem as if a focal lesion as such might cause a transitory mental disorder. But these cases are difficult to analyze and one should be careful with the frequent statement that a "little hemorrhage" or "embolism" is the cause of a transitory mental disorder.

As a rule the weight of the brain is very much reduced in old age and this reduction keeps pace with the dementia, so that in the simple delusional forms and in many cases of senile melancholia, we find the brain weight relatively high, whereas actual *dementia* is accompanied by a fairly parallel loss of weight. In many cases the atrophy refers more especially to special localities, such as the temporal lobe, and without there being any focal lesion whatever, of the nature of blocking of circulation or softening, we can obtain fairly pure symptom-complexes of aphasia in such cases with local atrophy. Even in these cases it is not always easy to demonstrate a parallelism between the atrophy of the part and the condition of the blood vessels feeding it. It is impossible to draw a line between the cases of simple atrophy of the brain and those of atrophy with focal lesions, except where lesions implicate a part which we can recognize by its focal symptoms during life. In other words, the vascular nature of the dementia can be diagnosed only on the existence of distinctly vascular symptoms.

As a rule the vascular lesion affects chiefly the vessels of the base; at times, however, it extends into the smaller branches and very diffuse changes are possible, such as multiple small aneurisms and hemorrhages, not only in the place of predilection (the internal capsule) but in various parts of the cortex. One of these patients had affection of the cerebellar vessels as well as those of the cerebrum, and the condition of ataxia produced

in this way had erroneously led the committing physician to the diagnosis of locomotor ataxia, which was easily excluded owing to the presence of exaggerated reflexes referable to the affection of the motor area and pyramidal tracts. The difficulty of distinguishing such cases from general paralysis is at times great, especially in the absence of a history of the development.

Among my autopsies I make the following groups:

1. Cases with focal lesions such as softening, hemorrhage, or temporary ischæmia, or focal simple atrophy, causing hemiplegia, aphasia, or hemianopsia, demonstrable during life with varying degrees of general atrophy and dementia.

2. Cases with foci of softening which gave no focal symptoms. I mention here especially the numerous small cysts which occur in the corpus striatum or in the centrum ovale, many of which may make no symptoms whatever, whereas others may encroach on the pyramidal tract and, when they involve the anterior limbs of the internal capsule, not infrequently bring about a peculiar condition of irresistible crying or laughing, which practically allows a diagnosis of this focal lesion.

3. Cases with multiple very small foci of softening, especially of the cortex; several of these cases developed epileptic attacks during the last years of their life.

4. In other cases the lesion is fairly uniform and diffuse: usually a hardening and uneven shrinkage with overgrowth of the neuroglia, pigmentary atrophy and disappearance of whole nests of cells, widening of the ventricles and formation of so-called arachnoid cysts, owing to the shrinkage of the brain substance.

Therapeutically little is to be done beyond what all mental disorders require; provision for safe and simple conditions of life, the avoiding of encumbrances in the intestinal tract, and of states which naturally increase the blood pressure, such as worry and anxiety.

The principal value of our knowledge of the disorder is undoubtedly the prognostic one, and that of early recognition of danger in business transactions, and naturally also the avoidance of confusion with syphilitic processes, which would demand specific measures.

VIII. SYMPOSIUM ON ARTERIO-SCLEROSIS

7. Pulsus Infrequens

By THOMAS E. SATTERTHWAITE, M. D.

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In looking over the literature of this subject lately, I found no less than thirty-eight American physicians had contributed to it. Perhaps many more have done so. And foreigners have added their proportion. So that it is evidently a subject that has created a wide-spread interest. And it is bound to increase until we have mastered the essential facts in its causation, natural history and treatment.

By way of introduction, I may say that the prevailing terminology of this affection is unfortunate. I refer to the words Bradycardia[1] and Spanocardia that are in use, and Araiocardia and Oligocardia, that have been suggested.

The use of the termination *cardia*[2] implies that the key to the actions of the arterial current is to be found, from a clinical point of view, in the heart, rather than in the peripheral arteries. And yet a comparison between the readings of recording instruments that register the heart and pulse beats simultaneously, as shown by Mackenzie[3] and others, illustrates that there may be a wide variation as to frequency and rhythm between the two.

To illustrate, a double contraction at the left ventricle may be registered in a sphygmogram, as a single one; while a cardiogram will show cardiac contractions of which there are no traces in the sphygmogram. This latter circumstance, characteristic of the "intermittent pulse" (to be distinguished from the "deficient pulse" where both cardiac contraction and the pulse beat are simultaneously "missed") is of course very common. Now though the heart regulates the general circulation, its essential characteristics are better exhibited at the periphery than at the center. And for two reasons. The peripheral arteries are more accessible, and therefore can be more conveniently studied than the heart, and give us more information because cardiac action studied at a distance, can be better appreciated. It is not uncommon to judge of motive forces by their remote effects. In telegraphing or telephoning, the one who transmits the message cannot judge of the quality of his instrument, so well as the one who receives it. Clinically, therefore, the circulation is bet-

ter estimated by the pulse than by the heart. So that there is reasonable objection to the use of all terms ending in *cardia*. But apart from this, the qualifying prefixes are either improper as in the use of *brady*, or are vague in meaning. True *Bradycardia* implies that the ventricular contraction is prolonged beyond the usual time, that it is slow, in other words; but so far as I know, slow ventricular contraction occurs only in aortic stenosis or in aneurisms near the aortic orifice, where some obstruction like an atheromatous plate narrows the lumen of the vessels, the contraction of the left ventricle being necessarily prolonged, in order to permit it to force the column of blood through the constricted passage.

But this is not the common acceptance of the term, which is simply that the number of pulse beats to the minute is abnormally small. As a matter of fact, however, pulse beats, in the infrequent pulse, may be quick or slow.

To my mind of all the terms suggested, no one expresses this numerical deficiency so well as the Latin *Pulsus Infrequens*, the infrequent pulse.

The pulse rate, as is well known, varies more or less according to circumstances, such as the age and height of the individual, atmospheric temperature, the time of day, acquired or inherited peculiarities. In the adult male the standard is set at seventy-two; in the adult female at seventy-six to eighty. And yet it is by no means rare for a person of apparently good health to have a pulse anywhere in the sixties. By general consent, however, a pulse below sixty is to be regarded as infrequent.

The *pulsus infrequens* may occur at almost any period of life. Prentiss[4] has reported one instance at sixteen months. It was caused by an injury to the neck, followed by an abscess between the medulla and pons.

Grob, from elaborate study, says it may occur as late as ninety, usually, however, between twenty and forty. In my experience it is more often seen in the middle period of life, or after it.

The infrequent pulse in adults (where the rate falls below forty) is rare, though most practitioners with large experience have probably seen occasional instances at some time or other in their practice. If, however, we should adopt sixty as the figure below which all pulses are to be reckoned as infrequent, we would find them comparatively common. According to Grob's experience about one individual in forty[5] has an infrequent pulse. Thus far it has been unfortunate, however, that most observers have failed to note the relation in number per minute between the pulse and heart beats, though the importance of ascertaining this variation was pointed out by Stokes[6] in 1846, when he told

of a patient whose heart beats were thirty-six to the minute while the pulse was twenty-eight. Since that date the importance of this point has been emphasized sufficiently to have merited general attention.

Four different relations may be observed between the action of the pulse and the heart: (1). The heart and pulse may beat simultaneously. (2). The heart beats may not all be communicated to the wrist. (3). The auricles may pulsate more frequently than the ventricles and arteries. (4). Contractions of the several chambers may occur, when the heart has been removed from the body; and even when the muscular tissue of the walls of the heart has been cut into small pieces, they may be made to contract by various stimuli,—a fact that has been known since the time of Vesalius.

The first two of these statements have been satisfactorily proved by clinical experience; the last two by physiological experimentation, where electrical currents and other stimuli have been known to produce muscular contractions hours after death.

The infrequent pulse has two principal varieties, the physiological and the pathological.

Of the first we have two well-known instances, the infrequent pulse of inheritance, and the pulse of pregnancy.

Prentiss has recorded several instances where persons whose pulses averaged thirty to thirty-two were in apparently sound health, and historians tell us not only that Napoleon's pulse was forty even in the midst of a battle but that he felt uncomfortable when it rose to sixty. The most remarkable instance, however, is, I think, that of Vigouroux[7] who had under his observation a laborer, whose pulse never exceeded twenty. The man never experienced any illness so far as he knew, except on one occasion when he had a short and slight gastric attack, that was successfully treated. In this connection it is interesting to remember that Czermak[8] could stop the action of his heart for a few beats by pressure on the pneumogastric and Quincke[9] has verified this experiment. Besides, the heart has been stopped by stopping the respiration.

The infrequent pulse is more common in males than in females, the ratio being about five to one according to Prentiss's tables.

Of the pathological we have two subdivisions, the Paroxysmal or Periodic or Temporary, and the Chronic or Essential. Under the causes of the former come the infections, typhoid, diphtheria, pneumonia, erysipelas, puerperal affections and influenza, best seen during convalescence, in toxæmias from lead, tobacco, tea, coffee, digitalis, uræmia, cholesteræmia and syphilis, in func-

tional nervous disturbances, reflex influences from the skin or gastro-intestinal tract, and in temporary debility.

Under the pathological variety are organic diseases of the brain or cord. According to Prentiss the infrequent pulse is chiefly due to organic disease of the brain or cord, epilepsy and organic heart affections, though in eighty-nine of his cases the cause was stated to be unknown in thirty-five.

Grob in his etiology, based on personal observations, is quite definite on this point. In a series of 100 cases he has put the etiological factors as follows, as to relative frequency:

Physiological	6
Idiopathic	1
Articular Rheumatism	24
Circulatory Disturbances	1
Digestive Disturbances	10
Diseases of the Central Nervous System	6
Infections and Constitutional Diseases	9
Convalescence from typhoid especially, but also diphtheria, measles, pneumonia, and erysipelas	43
	100

The paroxysmally infrequent pulse according to Grob represented 112 out of 140 of his cases. His experience that the paroxysmally infrequent pulse largely predominates coincides with my own views. The pulse rate in these instances under pathological conditions is subject to wide variations in range while, as we have seen, under physiological conditions, it maintains a tolerably steady rate. In the first named condition, very low rates have been recorded. Holbertson has published one instance where the pulse fell on one occasion to seven and one-half in a patient who had attacks of vertigo and loss of consciousness following an accident on the hunting field. At the post mortem examination it was found that there had been pressure on the medulla and upper part of the cord, the result of fracture of the occipital bone and upper cervical vertebræ. Bony union of the parts had ensued but with displacement of the fragments. Other instances have been published where the pulse fell as low as to four[10] and even three.[11] This last case was one of the paroxysmal variety, and the patient rallied from the attack.

The explanation of the infrequent pulse is not simple. We can realize that pressure on the pneumogastric may cause it, as in Czermak's and Quincke's experience, and there are many where there was organic disease at the base of the brain from pressure by bone, as in Holbertson's case.[12] This nerve passes from its root in the medulla down to the cardiac plexus. The augmentor (accelerator) fibres of the sympathetic also pass

down from near the same spot to the cardiac plexus, supplying energy to the muscular tissue of the heart, though the precise course of the fibres is unknown. Possibly they pass down through the cord.

The pneumogastric regulates, *i. e.*, moderates or restrains the energy of the heart, thereby showing up the pulse.

The use of the intrinsic ganglia in the heart substance is not yet understood. They may supply motor energy to the heart, independent of the spinal nerves or the sympathetic. And yet they appear to lose vitality after separation from the peripheral nerve system. Certainly the heart beats in the fœtus before any trace of nerve fibres can be found. Lastly it appears, that the muscle tissue of the heart may assume the initiative, acting independently of any nervous influence, and recent experiments appear to prove it. The paroxysmal cases may be due to reflex excitations of the pneumogastric, though the stimulus of almost any afferent (sympathetic) nerve, (such for example as the abdominal sympathetic), may cause them. For a blow on the abdomen conveys the impulse to the medulla through the pneumogastric, slowing or stopping cardiac action. These attacks may also be due to depression of the augmentors, such as occur in nervous or muscular strain, and in gastro-intestinal irritation. They may also be caused by diminished action of the accelerators. In the permanently infrequent pulse we appear to have these causes in continuous action.

There may or may not be symptoms. In the physiologically infrequent pulse, such as the hereditary, or congenital, or pulse of pregnancy, here are no untoward symptoms. In fact, evidence goes to show that in most of them, or certainly in many, an increase in rate begets disagreeable sensations.

On the other hand, the infrequent pulses of the infections, such as typhoid and the toxæmias, poisoning by tobacco, digitalis, tea or coffee, uræmia and anæmia, cholesteræmia and syphilis, are so wrapped up in the symptomatology of their several affections that a description of their several symptoms would carry us beyond the scope of this short paper.

We have now to consider the remaining forms of the neuroses (the so-called idiopathic varieties), a definition which, from a theoretical point of view, fits them conveniently enough, because they may turn out to be due to reflex excitations, as from gastro-intestinal disturbances, or skin diseases, or to direct excitations as after severe muscular exercises or nervous strain. In these latter cases the symptoms are those of general lassitude, prostration, præcordial oppression, the sense of constriction, choking and dyspnœa, are apt to be associated with nervous strain.

On the other hand, the results of severe muscular exercise are apt to be reflected on the nervous system by attacks of vertigo, convulsions, unconsciousness, of short or long duration, epileptiform or apoplectiform seizures, loss of rhythm, a systole associated with varying rates of infrequency of pulse, Cheyne-Stokes respiration and a synchronous pulsation of auricles and ventricles, which come on without warning or with an ill-defined aura.[13] This latter variety has now been erected into a group under the name of the Stokes-Adams syndrome,[14] because these two men were the first to describe it. Cases of this variety may survive for several years.

I saw such a case many times in consultation in 1901 (Case 1). An old gentleman with arterio-sclerosis, during a prolonged attack of heart failure, with a pulse in the thirties, while the heart beats were not far from normal, developed orthopnœa and ascites and had Cheyne-Stokes respiration during a period of unconsciousness that lasted over a week. But recovery took place and now, after the lapse of about two years, his physician reports that he is in fairly good health, driving in the park in good weather, and going to the theater when he feels inclined to do so. Digitalis was used in small doses for the ascites for brief periods only, the treatment otherwise being symptomatic.

On the other hand, there is a group that seem to be dependent on gastro-intestinal irritation, and its signs are flatulence, acid dyspepsia, nausea and vomiting.

The diagnosis offers no difficulties, but we should never in these cases rest satisfied until we have examined the heart to find whether it beats synchronously with the pulse. The simultaneous use of the sphygmograph and cardiograph may be instructive in these instances. And we should carefully run over the etiological causes, finding out first whether it is physiological or pathological, and if the latter, whether it is not merely one of the symptoms of the several diseases that have been enumerated. Of the prognosis but little can be said because the details are very scanty. In the physiological variety it is favorable so far as we know, but the instances are few, and the expectation of life has not yet been worked out.

In the paroxysmal forms, the prognosis depends on our success in mastering the disease, of which it is a symptom, and each affection gives a different expectation. For example, the infrequent pulse of digitalis or tobacco ceases when these herbs are withdrawn; in the infections it ceases with established convalescence; but in chronic diseases like lithæmia, uræmia, syphilis, a good outlook can only be assured when these diseases are held under firm control. Now if the physiological variety together

with the paroxysmal represent, as according to Grob they appear to, more than eighty per cent. of the cases, the prognosis is on the whole favorable.

On the other hand the permanently pathological form has a less favorable prognosis.

In the treatment no greater mistake can be committed than in aiming to accelerate the pulse by medication simply. Experience has abundantly shown that such treatment has invariably bad results. The physiological cases require no special treatment, and even the pathological appear to do best when let alone so far as the frequency of the pulse is concerned.

Especially when the infrequent pulse is associated with a recognized affection or condition like typhoid or lithæmia or syphilis, gastro-intestinal irritation or skin disease and anæmia, the treatment apposite for the underlying affection will affect the pulse favorably, and no special medication of the infrequency is necessary. Even alcoholic or diffusible stimulants should be used with caution. The nitrites and bromides are safer but it is always better, so far as possible, to treat the underlying disease without special reference to the infrequency of the pulse.

When, however, we are inclined to suspect that the infrequent pulse is due to a functional nervous disturbance, or are in doubt as to its exact cause, sedatives like the monobromate of camphor, assafœtida, valerian and Hoffman's ether are the remedies par excellence, together with carbonated baths and resistance exercises, carefully regulated diet, and observance of the sound rules of health.

The following cases taken from my pathological records and private practice illustrate the graver forms of the infrequent pulse:

Case II. Temporarily infrequent pulse due to digitalis. C.; age forty-four years. For five years had suffered from rheumatism, and for eighteen months from dyspnœa and palpitation. One week before admission to hospital his feet began to swell, and on admission he was found to be othopnœic. Urine contained twenty per cent. of albumen by bulk. After treatment by digitalis, in half ounce doses of the infusion, his pulse fell one day to thirty-six, when the digitalis was stopped. On the next it had risen to thirty-eight, and three days later to forty-five to fifty-four. Compressed air was then given and pulse reached eighty-four. Digitalis was given subsequently, but the pulse having fallen to fifty-two, it was stopped. About two and a half months later the patient died from rupture of the chordæ tendineæ of the mitral valve (it was thought). The aorta contained extensive atheromatous plates. The heart weighed thirty-two ounces. The infrequent pulse was ascribed to digitalis.

Case III. Temporarily infrequent pulse; mitral stenosis; chronic nephritis; ascites, treated by digitalis. J.; age thirty-seven years. Three

years before admission to hospital he began to have palpitation, dyspnœa and præcordial pain, and three months previously œdema of the feet, the urine being reduced to twenty ounces.

On examination no radial pulse was found. Extreme dyspnœa. Cough and expectoration. Murmur in mitral area. Under the infusion of digitalis in half ounce doses improvement ensued, but after two weeks' use of it the pulse began to be infrequent and the general condition worse. After the digitalis was stopped the patient's condition improved and with it the pulse, but, becoming irregular again, it was resumed, carbonate of ammonia and spiritus frumenti being added to each dose. The improvement in the pulse was only temporary, for one month after admission it had fallen to forty. During the summer the patient was absent from the hospital, returning in the autumn with no radial pulse, irregular cardiac action, and urine reduced to twelve ounces per day.

From this second attack the patient did not rally. At the post mortem examination the heart was found to be fatty, hypertrophied and dilated, weighing twenty-two ounces. The mitral had a button-hole opening. There was also chronic nephritis.

Case IV. **Temporarily infrequent pulse; mitral disease; chronic nephritis; abdominal dropsy.* A lady from Louisville, Ky., came under my care in the Post-Graduate Hospital recently with pulse 64, temperature 98, respiration 24. She had been under pretty constant medication by digitalis and opiates. During her stay in the hospital there were temporary suppression and subsequent lobar pneumonia. On four occasions she had attacks of heart failure, the pulse registering once twenty-eight, and three times forty-four. She recovered under the use of nitrites and alcoholics with occasional use of digitalis for diuretic effects only. On her return to the South the pulse had risen to seventy-six.

Case V. Mitral stenosis; phthisis; infrequent pulse. In still another case of mitral disease seen in consultation, where the patient had suffered from hæmoptysis and had suppression and albuminuria, the radial pulse ranged from thirty-seven to forty-four and was intermittent and irregular. Though I have learned that both cases III and IV are now dead, I am not aware that any post-mortem examinations were made.

The following cases illustrate lesser degrees of infrequency, when the rate falls below the standard, viz., sixty:

Case VI. Infrequent pulse in general tuberculosis and tubercular meningitis. S.; age nineteen years; stable boy. Six months before admission to hospital he had caught cold, as he expressed it, and since that time had lost twenty-five pounds. He complained of pain in head and a stiff neck. Pulse 42, temperature 100, respiration 20. Dry tongue, scowling brow, abdomen retracted. Albuminuria. After five months' stay in hospital patient suddenly became delirious. Pulse weak, 52. A month later, with temperature 99.5 and respiration 24, the pulse had risen to 64. But after another month it had fallen to 52. About a year later the temperature became subnormal (97.5), respiration 24, pulse 60. At the post-mortem examination tubercular pericarditis was found. In the lungs, liver and

* Previously reported by the author.

kidneys and at base of brain miliary tubercles. Cerebral ventricles distended with serum.

Case VII. Melancholia; temporarily infrequent pulse. Z., a young man listless and feeble, complaining of pain about the head, spine and chest, with a previous history of glycosuria, unable to apply himself to any intellectual work, was under my care in 1898-'99. Lateral curvature with protrusion of right scapula. Misshapen head, marked difference between the lateral halves. Left exophthalmos. Pulse sixty to sixty-four. Under treatment by carbonated baths and exercises, the pain in the back disappeared, the pulse became more frequent so that from an average of sixty it reached seventy-two. After leaving my care for the summer the patient relapsed into his former condition, but after a second course of treatment the pain in the head disappeared and the pulse again improved. The infrequent pulse was attributed to his neurotic condition, the symptoms being those of chronic basilar meningitis.

Case VIII. Mitral stenosis; periodically infrequent pulse. I have under my care now a patient who came to me in August, 1899, suffering from præcordial oppression, dyspnœa and a dilated heart, with feeble impulse, weak and irregular action, rate fifty-two. Presystolic murmurs over the mitral area, not conveyed. Occasional thrill. After treatment by carbonated baths and resistance exercises with strychnine, his pulse rose to the normal. His heart has now nearly regained its normal dimensions; there is absence of præcordial pain and dyspnœa; and the patient is leading an active life as the member of a very prominent firm in this city. His pulse rate is sixty to sixty-four.

Case IX. Infrequent pulse in uræmia in association with diabetes and mitral disease; temporarily infrequent pulse. A lady of middle age, the wife of one of our most distinguished practitioners, was placed under my care some years ago. As a child she had suffered from inflammatory rheumatism, and later from sciatica, getting relief from atropia and morphia. When first seen she had albuminuria; sugar in large amount, and some suppression. Vertigo, pain alternating between the occiput and vertex, dyspnœa. Pulse fifty-six, intermittent. Heart's sounds almost inaudible, but on moving rapidly about in her room a slight murmur conveyed to the left was detected.

The uræmia, and with it the infrequent pulse, was in this instance promptly overcome by the use of the muriate of pilocarpia, combined with alcoholics. According to my experience this drug may be given in divided doses up to as much as one and one-half grains per day, if guarded by alcoholics and watched by a competent nurse.

In the following case the infrequent pulse has a chronic character:

Case X. Arterio-sclerosis; apoplectiform attacks; lithæmia; infrequent pulse, at first periodic and later chronic. A patient in middle life came under my observation in consultation many times in April of 1901. In the preceding month he had an apoplectiform attack followed by stiffness in his knees and hips, and a sense of general lassitude. The usual local applications were made and in addition he took twenty grains of aspirin four times a day and in a few days was relieved of his pains; but muscular

inability of the left masseter remaining, he took salophen in twenty-grain doses every three hours, but was obliged to return to aspirin for relief of his pain. At this time the pulse had fallen to fifty. When I first saw him he was taking morphia in small quantities, as he had taken it previously for rheumatic attacks. I immediately stopped the latter, putting him on one-fortieth grain doses of sulphate of strychnia together with half-ounce doses of whiskey every four hours, and a liquid diet. Pulse was forty, but full and regular, synchronous with the heart. Respiration twenty-two to twenty-four. Under this treatment the pulse had risen, on May 5th, to sixty. Digestive disturbances causing intermittence of the pulse, strychnia was diminished and caffein in one-grain doses with assafœtida three grains were substituted, while ocasional doses of nitro-glycerine, one one-hundredth of a grain, were added. Under this treatment the pulse continued to rise so that on May 6th it was sixty-eight. But being deficient in force, digitaline (Merck) was given on one occasion four times during the day, nitro-glycerine being substituted for it in doses of one one-hundredth of a grain every fifteen minutes, whenever necessary. During all this time about two and one-half ounces of whiskey were being given daily and after this nitro-glycerine and strychnia as required with whiskey at intervals of about three hours. On May 10th, without obvious reason, the pulse had again fallen to forty-one to forty-two, but, as it had force and regularity, no effort was made to increase its frequency. For about five days it remained at forty, being strong and regular. Later arsenious acid in one one-hundredth grain doses and cactus in the fluid extract form in one minim doses were given, the pulse gradually rising under them to sixty. On May 17th it had reached seventy. The sulphate of quinine in eight-grain doses and morphia in one-eighth grain doses for relief of the pain replaced the arsenic, but the cactus was given with interruption, and with no other remedy for a week, when strychnia was again given. The pulse then returned to its normal frequency and continued there, although on a single occasion it fell to fifty-two. On June 26th the patient was suddenly attacked with pain in his left arm and wrist, which became swollen, the pain recurring every morning for five to six days. The pulse fell to forty-five. On the morning of July 5th the patient was awakened with buzzing in his ears, great dizziness; no pulse at the wrist. Aromatic spirits of ammonia, an ounce of whiskey and a dose of nitro-glycerine, one one-hundredth grain, were now taken and relief was obtained in about half an hour. This attack, however, was so prostrating that the patient was confined to his bed with it until September 6th, the pulse ranging from thirty-five to forty-four. The treatment during this time was by strychnia one thirtieth grain, nitro-glycerine one hundredth, and whiskey one half ounce, taken every three hours. The suprarenal extract in three-grain doses was given for a week, but did not sustain the pulse and was therefore abandoned. Some improvement in the pulse, however, was always noted after the use of the static battery. It would rise to about fifty on these occasions. On the 23rd of November the pulse reached sixty, when all remedies except whiskey were suspended. During the last year the pulse has averaged between forty-eight and fifty-four, the remedies used being strychnia, nitro-glycerine, and suprarenal extract, increased to five grains, three times a day. During the present month I have examined him with

the following results: Pulse thirty-six to forty-four, of fair force and regular. Heart beats synchronous with the pulse. No enlargement of the heart. No displacement of the apex; no murmurs, but impulse lacking. No thrill. Accentuation of the second sound at apex. No palpitation. Liver a little enlarged; some chronic bronchitis. Physical condition fairly good; takes no remedies; eats well, sleeps well and walks from one to two miles a day without fatigue or embarrassment of respiration, but he has the facies arterio-sclerotica.

This interesting case has now entered upon the chronic stage. By some it would be called an instance of permanent bradycardia. I should prefer to call it the chronic form of the infrequent pulse.*

Attention is called in conclusion to the following points:

(1.) The importance as bearing on prognosis and treatment of determining whether the infrequent pulse is physiological or pathological;

(2.) And whether this pulse is merely intermittent or deficient;

(3.) The association of this condition (as shown in the cases cited) with mitral disease, (stenosis especially) arterio-sclerosis, brain disorders, diabetes, chronic nephritis and poisoning by digitalis;

(4.) The danger of giving digitalis in large doses and the fact that milder cardiac remedies to the almost total exclusion of digitalis or its congeners may alone or in conjunction with hydrotherapy, resistance exercises, massage, electricity and compressed air be effectual remedies;

(5.) The inadequacy of our knowledge as to the causation of the infrequent pulse; and finally,

(6.) The inadvisability, if not impropriety, of using the terms of Greek derivation ending in *cardia* as synonyms of this condition.

1. Introduced by Grob. Deutsch. Archiv für klinische Medicin, 1888, XLII., p. 574.
2. Kardios, heart; brados, slow; spanos, deficient; araios, rare; obligos, few.
3. Mackenzie. Study of the Pulse, Edinburgh and London, 1902
4. Prentiss. Transactions of American Physicians, 1839, p. 120.
5. Grob. 82 in 3578 patients.
6. Stokes. On the Heart, etc., 1855, p. 329.
7. Vigouroux. Gazette des Hopitaux, 1876, p. 788.
8. Czermak. Viertel Jahresch. für pract. Heilkunde, 1868, p. 120.
9. Quincke. Berliner klinische Wochenschrift, 1875, No. 15, p. 190.
10. W. Henry Day. British Medical Journal, 1880, vol. I., p. 118. The pulse beat four times a minute for about four minutes during an attack of unconsciousness.
11. Prentiss. In case seventy-nine.
12. Holbertson. Medico-Chirurgical Transactions, vol. XXIV., p. 76.
13. His, Jr. Deutsch. Archiv für klinische Medicin. Vol. LXIV., p. 816.
14. Huchard. Mal du Coeur, Paris, 1898, p. 309.

* This history has for the most part been prepared for me by the patient himself.

DISCUSSION

Dr. E. LIBMAN, of New York: The time at my disposal is so short that I shall refer only briefly to the points to be made. I shall include all of the forms of chronic disease of the arteries under one head. The most important thing perhaps in discussing the significance of arterio-sclerosis is the question of location. When these cases are examined post-mortem one finds a simple atheroma at the attachment of the aortic valves to the aorta, or, without involvement of the aortic valves, there will be nodules around the orifices of the coronary arteries. Sometimes the process extends throughout the aorta, and sometimes the abdominal aorta is more involved than the thoracic aorta. There is a curious fact which has been observed by a number of pathologists, *i. e.*, the frequency of the occurrence of marked atheroma of the splenic artery and of aneurism. The exact significance of this is not known.

As regards the etiology I would say that it has been pretty well proved that obstruction of the circulation does undoubtedly produce arterio-sclerosis. The most marked case I ever saw was in connection with congenital stenosis of the aorta. The same has been observed in cases of atheroma of the pulmonary artery occurring in connection with mitral stenosis. There are on record a number of cases of primary atheroma of the pulmonary artery extending into the smaller branches which give the general picture of cardiac disease, and cannot be diagnosed.

The atheroma is not infrequently seen in young people. Cushman, for instance, has found sclerosis and aortic aneurisms six times in persons under twenty-five, and atheroma has been seen in children. At one time I published with Dr. N. E. Brill such a case, occurring in a girl of sixteen. Recently a case of calcification of all the arteries of the body of a child was reported. I wish also to call attention to the value of the examination of the arteries of the forearm and lower extremities in making the diagnosis of arterio-sclerosis. There may be extensive calcification of the peripheral arteries without any involvement of the aorta proper. It is important in examining patients not only to examine the radials but also the brachials, because for the early diagnosis of atheroma of the arch of the aorta Cushman has pointed out that search must be made for the difference of the pulse on the two sides. Experience has shown that it is not safe to look for this only in the radials.

I should like to refer to the question of œdema of the lungs occurring in persons with arterio-sclerosis. It has been believed that such a condition can occur without stagnation, simply as a result of some disease of the aorta and coronary arteries. Most of us, I think, have seen such cases occurring in persons with arterio-sclerosis and myocarditis; they have developed pulmonary œdema without other assignable cause. About one year ago I had a patient who had repeated attacks of pulmonary œdema, the attacks terminating with fluid discharge from the bowels. The pulse remained good throughout the attacks, and the latter were controlled by morphia.

Finally, I wish to call attention to the occurrence of disease of the vessels in the lower extremities of young people, particularly those between thirty and forty years of age. It is noticed more particularly among Russian and

Polish Jews, though the reason for this has not been ascertained. These cases often present a long history, and often an erroneous diagnosis, such as rheumatism, is made. In these cases the lesion in the vessels is somewhat different; it is a pure obliterating endarteritis with thrombosis above or below the point of narrowing of the vessel. I have had occasion to make two autopsies in cases of that sort, and in both instances there was absolutely no disease of the other vessels of the body.

As to the question of the relation of infections to arterio-sclerosis, I would say that there is a certain amount of evidence to support this view, but not enough attention has been paid to the significance of infections in persons with arterio-sclerosis. These people are particularly liable to develop systemic infections. They are especially liable to acute endocarditis, and the thickened valve here becomes just as much a point of least resistance as the thickened valve does in acute rheumatism. Not only the valves, but even the aorta itself, may become involved. When patients with aneurism develop an elevated temperature we should think of the possibility of a superimposed infection.

Dr. A. L. Benedict, of Buffalo: It occurred to me that if we could use the word apparatus where "system" is used on the program we might obtain some hint as to this principle. An apparatus is some local accumulation of special organs for a special purpose. In discussing the relation of changes in the vascular system to the digestive apparatus we must consider the three general principles: (1) General pathological changes bearing a direct causal relation to lesions in an apparatus; (2) lesions of the two which are purely coincidental, and (3) relations of the general change to the local. Except in a strict histological sense I do not believe there is any change of the digestive apparatus that may not be directly due to arterio-sclerosis, and, on the other hand, that any one of these conditions may be due, and is in the majority of instances due, to any other trouble.

With reference to baths, I would say that the introduction of hot air baths in arterio-sclerosis is like the introduction of exercises in heart disease. Anything which will exercise the arteries and change arterial tension is contraindicated, but in other stages the baths are indicated, not only because of their eliminative action, but because of the exercise of the vessels. In hepatic sclerosis I think we have paid too much attention to the portal vein and to alcohol acting through this vein. There are a good many other poisons than alcohol causing cirrhosis, and we may have a form of hepatic cirrhosis due to general poisons reaching the liver through the hepatic artery, often coincident with, or perhaps really essentially an, arterio-sclerosis of the hepatic artery and its branches. Embryologically, the liver in relation to the hepatic artery and the kidney in relation to the renal artery, occupy very similar relations. These arterio-sclerotic changes may have, I think, a great deal to do with hepatic cirrhosis.

Dr. Edward D. Fisher, of New York: I should like to refer to one or two points in connection with this very interesting discussion. In atheromatous disease of the vessels I think we can look upon the endarteritis as being general, for, I do not think any of the arteries are liable to escape. However, when we come to cases of syphilis we are not dealing with a

general and ordinary endarteritis, but one affecting exclusively or specially the vessels of the nervous system. This gives rise to thromboses and hemorrhages in the brain, and to disease of the vessels in the spinal cord, giving mixed spinal paralyses. In another class of cases there is apparently no history of either syphilis or of the toxæmias or of kidney complication. When apoplectic seizures occur in the comparatively young I would place the cause as some very great strain associated probably, not with endarteritis, but nevertheless with a disease affecting deeply the muscular coat of the arteries. Syphilis, alcoholism and kidney complications may be excluded in the class of cases which I have in mind.

Dr. HENRY L. ELSNER, of Syracuse: The question of the Stokes-Adams phenomenon has interested clinicians very much of late, and I rise to corroborate the statement, made by Dr. Satterthwaite, in connection with this part of the subject. These cases may continue for many years. For instance, I saw a patient about fifteen years ago who had what we then called bradycardia, the pulse running most of the time between eighteen and thirty. At times, the respirations were faster than the pulse. The condition continued between eight and twelve years. This case presented a pure Stokes-Adams phenomenon. There were repeated attacks and he finally died in coma after a number of weeks of slow pulse and increasing coma. On the other hand, there are cases of the Stokes-Adams phenomenon in which the course is very rapid. Two or three years ago I saw in consultation a man whose habits were excellent, and who was about sixty years of age. He presented the characteristic Stokes-Adams phenomenon. Death occurred in a few weeks. In both of these cases the arteries were diseased throughout, and in both cases there were marked evidences of myocardial degeneration. It is not the easiest thing to make the diagnosis of a true infrequent pulse. I think we should always use the sphygmograph if we fail to obtain exact data from the ordinary physical examination. This instrument has given, in recent years, some very interesting revelations, even after the careful study of the heart with the aid of the phonendoscope.

With regard to general and limited arterio-sclerosis, I think we should take a very conservative view. In some cases the clinician fails to palpate the arteries in cases in which one may be positive that arterio-sclerosis is present. Many cases of pure angina pectoris are met with where the artery at the wrist, and the other palpable arteries show nothing, and yet the patient finally dies of true angina pectoris after months of such attacks. Then there are cases giving no evidence of extensive arterio-sclerosis, and yet there is evidence of myocardial disease. The majority of these, I think, should be considered as of syphilitic origin, for, if we make a mistake the patient is given the benefit of the doubt. In the cases of erythromelalgia which I have reported there have been found far-reaching changes in the peripheral vessels.

Dr. ANNA M. STUART, of Elmira: I should like to add a case of gastralgia extending over twenty years, and finally terminating in an attack of angina pectoris. The attacks were so severe that they required nearly a grain of morphia for their control. Ten years before death disease of the

aortic vessels was diagnosed by Dr. Wey. During the four years the man was under my care, the attacks of gastralgia occurred regularly at two A. M. every other day for several weeks at a time. Under the advice of Dr. Allen Jones, of Buffalo, I gave the iodides, and they stopped the attacks for a short time. Six weeks before death the attacks of angina pectoris began.

Dr. JAMES J. WALSH, of New York: There is one thing which has perhaps not been brought up in the discussion, and that is the question of heredity. Physicians frequently find that families are practically wiped out at the age of fifty. If these persons are watched in early life the evidence of arterio-sclerosis will be detected. The real cause of it is a failure of what the Germans would call the original "anlage" of the arteries.

I saw one very interesting case of slow pulse in the person of a theater dancer whose pulse is never over thirty. It causes her no discomfort. She has had all of the usual diseases of childhood, and yet no one has ever told her that her pulse presents anything unusual. Apparently the slow pulse developed after an attack of the grip. She is able to carry her rather arduous duties without inconvenience.

Dr. DELANCEY ROCHESTER: With regard to the early occurrence of arterio-sclerosis I would say that three of the cases I reported were all under forty, and it seems to me that the reason we have considered arterio-sclerosis to be a disease occurring beyond middle life is that we have not recognized it earlier. This shows the importance of systematically investigating this subject at all ages. In reporting the cases I said that all *palpable* arteries were notably thickened. It is wise for us, I am sure, not to limit ourselves to the palpation of any particular artery or limb. One of the speakers said that a man of sixty-two died of congenital aortic stenosis, but I wondered how he made that diagnosis.

I omitted to mention in my paper the intermittent claudication, and I am glad it has been brought out in the discussion. The relation of mitral stenosis to arterio-sclerosis has not been brought out as I hoped it would be in the discussion of the heart symptoms. It is not at all uncommon to find mitral stenosis, particularly in women, associated with arterio-sclerosis. I have in mind four cases, three of which came to autopsy, in which there was distinct mitral stenosis. All of these patients were women at about the age of forty when I first saw them, but I do not know how long the disease had existed. Mitral stenosis is not always easy to diagnosticate without frequent examinations and careful study. Where the pulse is very slow and of high tension and small volume, we should suspect mitral stenosis and search for it most carefully.

Dr. STOCKTON: I have been delighted to find that the important subject of arterio-sclerosis still possesses interest, although it is difficult to say anything new upon it. It does seem to me that there is something yet to be learned about the relation of arterio-sclerosis to the digestive apparatus. There seems to be a good deal that is new in the literature on this subject. I am rather surprised that no one has mentioned in this discussion the work of Arthur V. Meigs, that rather remarkable book on "The Origin of Disease." In this book he gives histological examinations of a large number

of cases dying of different diseases, and shows that in many instances the persons really died of arterio-sclerosis.

Dr. SATTERTHWAITE: There is no question, I think, that arterio-sclerosis is hereditary sometimes. I have under my care at present a patient having arterio-sclerosis who also has hereditary ataxia, and two brothers are affected in the same way. Two other brothers have died of apoplexy. This man illustrates the hereditary tendency of this affection. Arterio-sclerosis may occur at any period of life, and we can only give information as to the results of our narrow personal experience. My own has been chiefly among people of about middle age. Arterio-sclerosis is by no means always general. If syphilis or lithæmia or uræmia is the cause there will be a general arterio-sclerosis, but those who have made post-mortem examinations will remember that arterio-sclerosis of the Sylvian artery at the base of the brain may be observed without any other artery in the body being similarly affected. It is most important that we should examine more than one artery.

With reference to treatment, I would say that I think the iodides should be given systematically for many months and even years to these cases if success is to be achieved. I have had a recent case of buzzing in the ears which has lasted for years, and which has been improved by taking for months the iodide of iron. The stomach often rebels against the iodide of potassium, but iodide of arsenic in tablet form in the dose of 1-100 of a grain is usually well borne, and does good. With regard to the hot bath, I would say that arterio-sclerosis is not, as a rule, successfully treated by the hot baths, especially when given, as in this country at night. They are apt to produce congestion of the brain, and are apt to make these patients irritable and cause them to suffer from insomnia. With reference to exercises in arterio-sclerosis I would say that they are apt to be followed by distress, and they are not advisable in the advanced cases.

IX. SYMPOSIUM ON HÆMATOLOGY

1. Blood Examination in General Practice

By IRVING PHILLIPS LYON, M. D.
BUFFALO

First, I wish to express my high sense of appreciation of the honor done me by the officers of your ancient and honorable body by their invitation to me, a non-member, to address you. Your president suggested that the treatment of the subject assigned to me be practical and suited to the interest of the general practitioner rather than the pathologist or specialist in hæmatology, for which suggestion I was glad, as it permitted me to speak at all on this occasion. For I had no new discovery to announce to the pathologist and, though this would apply as well to the practitioner of medicine, yet I trusted to his greater leniency of judgment, as at least I should be a no worse offender in such respect than most others who read papers at general medical meetings. Though, therefore, I shall say nothing that has not been said before, I shall try to convince some skeptic amongst you, perchance, of the real utility of blood examination in the daily practice of his profession. For, in spite of all that has been spoken and written on this subject, it is true that the profession, at large, still have only slight familiarity with blood examination as a method of diagnosis.

I have drawn from my personal experience, in private and hospital practice, the following few cases which I have selected to narrate, with all possible brevity, for the purpose of giving concrete examples of the value of blood examination in determining diagnosis and thus rational treatment, whether medical or surgical. By this I do not intend to imply that blood examination was the exclusive means of settling the diagnosis, but that it was an important, sometimes an essential, factor among all other possible means of information in determining the diagnosis. For all available means of obtaining light on a case were invariably adopted and the blood was merely one, though an important one, of such means. And I would expressly state my conviction that a blood worker of any merit must be an even better clinician than hæmatologist, neglecting no sign or symptom and carefully correlating all the facts of a case before reaching a judgment. For one person must ultimately make the

diagnosis and assume responsibility for it, and that person must combine the experience and matured judgment of both the clinician and the hæmatologist in order to properly correlate the whole evidence. The failure to recognize this principle has been the cause of much of the misunderstanding and discredit of blood reports and diagnoses bases thereon.

I shall limit my remarks to-night to citing only a few instructive cases, as examples, and to commenting thereon and, perhaps, on a few practical points of general interest in clinical blood examination.

Central Pneumonia, Obscured by Pleural Effusion.—This case occurred in my private practice in November, 1901, in a woman, Mrs. H., aged seventy-five years. The onset was sudden with chill and pain in the left side behind, following exposure to cold. The symptoms were those of pneumonia or acute pleurisy. The physical examination of the chest was almost negative and remained so until the end of the second day, when slight percussion dullness began to appear at the left base behind, without tubular breathing or râles. There was no expectoration. Next day friction sounds were heard over the relatively dull left base without other changes. On the fourth day flatness and positive physical signs of fluid were obtained and about 600 c. c. of clear serous fluid were aspirated. No tubular breathing or characteristic râles were then present. Was the case, then, one of simple acute pleurisy with effusion? No, it was a case of atypical pneumonia complicated by serous pleurisy, as made probable by the blood examination, which showed a high leucocytosis (35,000) even on the first day. The blood was examined daily and the leucocytosis persisted, with a relative increase of the neutrophiles, an iodine reaction in the leucocytes and a marked increase of fibrin deposit. I told the family, at first, my belief that the case was one of pneumonia, because the blood indicated it in spite of the negative physical signs. Not until the seventh day did tubular breathing and signs of pneumonic consolidation appear. By the next day, however, the process had extended and involved the entire left lung, which was solid and flat from top to bottom with intense tubular breathing and râles over it everywhere. The patient died on the eleventh day.

Was not the blood examination of value in this case? It is in cases in which pneumonia is suspected and cannot be determined positively by other means that the blood offers assistance. By means of examination of the blood, acute serous pleurisy and influenza can usually be differentiated from lobar pneumonia. Besides the usual leucocytosis, I have found a positive and marked iodine reaction in the leucocytes in all cases of pneumonia, and I have come to regard this reaction of distinct value in the diagnosis of atypical cases.

Pelvic Abscess.—Mrs. D., seen with Dr. Herman E. Hayd at the German Deaconess Hospital, in September, 1899, had developed a septic tempera-

ture two weeks after the birth of her baby. This had continued for about one month in spite of repeated uterine curetting and douches. It seemed that the symptoms depended on a further cause than the slight uterine inflammation with its free drainage and active treatment. The blood examination showed 40,000 leucocytes, a relative neutrophilia, an increase of fibrin and a marked secondary anæmia. The blood examination was distinctly in favor of abscess, and examination and operation a few days later located a pocket of pus between the uterus, bladder and omentum.

Perinephritic Abscess.—L. B., a boy of eight years, I saw with Dr. Lorenzo Burrows about two years ago. He had been running an irregular temperature with an occasional slight chill for a few days, and had been seen in consultation by several physicians. Various possibilities of diagnosis had been considered, especially typhoid fever, malaria, and tuberculosis. After a few days it was noticed that the boy lay in bed with his left thigh slightly drawn toward the abdomen and that deep pressure in the left flank caused him to wince. The urinalyses were negative. After about ten days of slight and indefinite symptoms, I was asked to examine the blood and found 33,000 leucocytes, the neutrophiles in excess, and a marked increase of fibrin. Upon inquiry as to the signs and symptoms of the case, I obtained the information already stated, and that appendicitis, pneumonia, empyema and pericarditis had been excluded, and further that nothing could be discovered except the slight tenderness in the left flank on deep palpation, the voluntary flexure of the left thigh and some discomfort to the patient when the thigh was actively or passively extended. In view of the blood picture, which was that of active inflammation, and of the localizing symptoms described, I made a diagnosis of "deep seated abscess in the left side, probably perinephritic." Two days later this diagnosis was confirmed by operation and by the discovery of about *half a teaspoonful of pus* behind the left kidney. The boy made a prompt recovery.

Who would deny the value of the blood examination in this case. Without it, days, perhaps weeks, might have passed before the diagnosis had become clear, and in the meantime serious damage to the kidney and other organs might have occurred.

Appendicitis.—Miss M. N., about nineteen years of age, had been taken with nausea and vomiting while at dinner two nights before my examination. Fever, a coated tongue and diarrhœa followed, with a little tenderness in the lower part of the abdomen, not sharply localized. Most of these symptoms subsided and the patient felt rather comfortable on the second day, a slight temperature, however, continuing. A very thorough examination of the appendix by Dr. D. W. Harrington, the physician in charge, and Dr. Matthew D. Mann, in consultation, at four o'clock on the afternoon of the second day failed to reveal any positive signs of appendicitis, and it was thought that appendicitis probably did not exist. For caution's sake, however, a blood examination was considered advisable, which I made in the early evening of the same day, with the result that I found 20,000 leucocytes, a relative neutrophilia, an intracellular iodine reaction and an increase of fibrin. The blood indicated appendicitis and, moreover, a well

advanced stage of the disease. An immediate operation was done and an appendix in an early stage of gangrene was removed. The patient recovered promptly.

What might have happened had this appendix been left alone over night or longer? The blood examination possibly saved this young woman her life. This case illustrates the importance of blood examination as a routine proceedure in doubtful cases of appendicitis. Physical examination alone failed to detect the disease, but blood examination was competent to show its presence. Such a single case as this one is sufficient answer to the scoffing criticism of blood examination in appedicitis that we have heard so persistently from a certain quarter.

In another case of suspected appendicitis that I saw recently with Dr. DeLancey Rochester, there were 6,600 leucocytes of normal differential distribution, a normal fibrin deposit, and an absence of the iodine reaction. In this case, on the second day the symptoms were highly suggestive of appendicitis. The blood was flatly opposed to it, however, and the patient promptly recovered without any further trouble. Many similar cases might be cited, but one suffices for illustration.

Intestinal Perforation in Typhoid Fever. I have had the opportunity recently of examining the blood in three cases of typhoid perforation in the Buffalo General Hospital, in the services of Drs. Charles G. Stockton and Charles Cary. In not one of these cases was a gross leucocytosis found, though a relative neutrophilia was present. In all three cases an intracellular iodine reaction was demonstrated. In spite, therefore, of the absence of leucocytosis, due to overwhelming septic infection by which the reactive power on the part of the blood is paralyzed, the regular presence of iodophilia may be of material assistance in determining the diagnosis. As iodophilia may occasionally occur in typhoid independently of any septic complication, its presence with symptoms of perforation is not a certain sign of perforation, unless slips of blood previously taken showed its absence before the advent of signs suggesting perforation. This caution indicates the method of avoiding error.

Acute Intestinal Obstruction.—This case was seen last July with Dr. Charles P. Chapin. The patient, Mr. G., aged about 50 years, had suddenly developed symptoms of acute intestinal obstruction and finally typical fecal vomiting. The bowels had not moved for several days and had resisted repeated efforts to secure their evacuation. Exploratory laporotomy was considered and almost determined upon. The blood was examined and found entirely normal. This was against any serious pathological condition of the intestine and encouraged a waiting policy. A renewed effort to move the bowels by large and repeated enemata proved successful at

last and was followed by a large number of passages of a surprising amount of fecal material. The patient was relieved of distress and made an immediate recovery. The blood examination was the chief factor in postponing and finally making unnecessary the proposed laparotomy.

Trichinosis.—Mr. B., a German, about fifty years old, I saw in consultation with Dr. E. R. Gould during last July. He had had a moderate temperature for a few days, felt rather sore in the calves of the legs, the eye-lids were slightly puffy and ecchymoses were noted in the conjunctivæ of both eyes. The blood showed 9,800 leucocytes, over thirty per cent. of which were eosinophiles. About two weeks before, the patient had eaten Westphalian ham, it was subsequently ascertained. As no other cause of so marked an eosinophilia could be determined after careful examination of the skin, stools, etc., it was concluded in view of the general symptoms that the patient was suffering from a mild trichinous infection. Unfortunately the opportunity was not afforded of confirming the diagnosis by examination of a bit of muscle from the calf.

Malaria versus Cancer of the Kidney.—In March, 1901, a woman about sixty years old was admitted to the Buffalo General Hospital, in the service of Dr. Roswell Park, for operation on what had been diagnosed cancer of the right kidney. A mass resembling the kidney could be felt in the right hypochondrium and the urine contained considerable blood. Moreover the patient had lost flesh, had grown weak and anæmic and had had chills. Malaria had not been suspected. A glance at the fresh blood preparation showed half-grown tertian malarial organisms, and there was a marked anæmia without leucocytosis. Upon inquiring, it was found that the patient had come from Georgia about six months previously, had had malaria in Georgia, and had had chills on alternate days for about two weeks before entering the hospital, with bloody urine following the chills. The diagnosis looked clear — *malaria, floating kidney, and hæmaturia.* Quinine was given, the chills and hæmaturia disappeared, the patient recovered strength and in a few days returned to her home, escaping an exploratory laparotomy.

Malignant Diseases of the Left Kidney versus Splenic Hyperplasia, Chronic Splenic Anæmia, etc. Every text-book of medicine lays down principles for the differentiation of splenic from renal enlargement, when a solid mass is encountered in the left upper abdominal quadrant. Such a differentiation is notoriously difficult in many cases, and any means of aid should be welcomed. I believe that such an aid is furnished by the blood. This has correctly guided me to a diagnosis in five cases of malignant tumor of the kidney, in four cases of chronic splenic anæmia, in one case of splenic hyperplasia secondary to alcoholic portal cirrhosis, and in one case of splenic enlargement associated with and probably due to chronic passive congestion of cardiac origin, although this case, too, may perhaps be one of splenic anæmia. I have met only one case in which the blood appeared to be a false guide, and this case is still living and may, perhaps, after all, accord with the rule.

The principle involved in the differentiation lies in the fact that malignant disease of the kidney is usually accompanied by a leucocytosis with relative neutrophilia, whereas, on the contrary, simple splenic hyperplasia rarely shows leucocytosis and often distinct leucopenia. Malignant disease of the spleen would agree with malignant disease of the kidney in its blood picture and might cause confusion, except for the fact that primary malignant disease of the spleen is so rare as to be almost a pathological curiosity, and may, therefore, be neglected in practice. Inflammatory mass of the kidney and large perinephritic abscess are more likely to confuse. As a rule, these conditions can be differentiated from cancer of the kidney by the history, symptoms, physical signs, urinalysis, and by the presence of the blood picture of abscess, *i. e.*, marked leucocytosis, increased fibrin, and iodophilia. Increased fibrin deposit and iodophilia together are not often found in the blood with malignant tumor of the kidney, though they may be if marked inflammatory degeneration involves the cancer of the kidney. Observing all these points, it is usually easy to distinguish abscess from malignant disease of the kidney. Leukæmia could cause no trouble, as a mere glance at a stained specimen of the blood would suffice to distinguish it, except in rare cases.

The problem is thus narrowed down to deciding whether a solid mass in the left hypochondriac region is a malignant tumor of the kndney or a simple non-malignant hyperplasia of the spleen, and the blood examination is usually competent to decide. This guide must be applied with caution and judgment and in strict conjunction with a careful weighing of the evidence furnished by the history, symptoms and signs of the case. Thus applied, it is of undoubted value, as evidenced by the following illustrative cases:

Cancer of the Left Kidney.—Case I.—Mrs. C., aged about sixty years, was referred to me by Dr. Matthew D. Mann in May, 1899, with the statement that she had a solid mass in the region of the spleen. The blood showed 17,000 leucocytes, of which 89 per cent. were neutrophiles, a normal fibrin deposit and a moderate secondary anæmia. I referred the patient back with the diagnosis, "cancer or inflammatory mass of the left kidney, probably cancer." The diagnosis of cancer of the kidney was fully confirmed, Dr. Mann subsequently stated.

Case II.—Mr. C., aged 39 years, was admitted to the Buffalo General Hospital, in the service of Dr. Roswell Park, in June, 1899, for operation on an "enlarged spleen of splenic anæmia." The tumor occupied exactly the splenic region, projecting downward and forward below the costal margin on the left side. It was firm and solid, smooth, non-movable, and had an anterior edge that was somewhat narrow or sharp, resembling a spleen. This mass had been growing rapidly for five months at least. No

blood had ever been detected in the urine. The patient complained chiefly of a soreness in the left side. The blood, taken on the morning of the operation showed as follows: red corpuscles, 3,804,000; hæmoglobin, 51 per cent.; leucocytes, 14,700; neutrophiles, 86 per cent.; fibrin, normal; an occasional normoblast and moderate poikilocytosis. With such a blood picture, I followed the rule and made a diagnosis of cancer of the left kidney, as opposed to splenic enlargement. Dr. Park, however, in view of all the circumstances, determined to explore the abdominal cavity. The abdomen was opened, the spleen was found normal in size, the wound was closed, the patient was turned over, the kidney was cut down upon and found involved in a large cancerous growth.

This case is particularly interesting, as the mass felt exactly like a spleen, even after the patient was under complete anæsthesia. Not a single point, except the blood examination, suggested kidney as opposed to spleen, and even the blood changes were rather slight. However, the operation showed that the blood, even in this case, was a truer guide than the physical signs. I might add that I have never encountered a case of malignant tumor of the kidney in which a hyperleucocytosis was constantly absent. However, such cases have been reported, though rarely, and their possibility must never be neglected.

Splenic Anæmia. On the other hand, the blood may be of distinct value in confirming the splenic nature of an abdominal mass in this region, as in the following case: Mr. G. S., aged fifty-nine years, I saw on February 21, 1899, in consultation with Dr. Carlton R. Jewett. A large mass with a sharp edge projected from the left costal border well down into the abdomen. For about one year the patient had been growing weak and anæmic. The blood showed a marked anæmia and only 4,800 leucocytes. A second blood examination, three months later, showed a more advanced anæmia and only 3,075 leucocytes. Two weeks after the second blood examination the patient died, and the necropsy showed a hyperplastic spleen of chronic splenic anæmia.

Simple Splenic Hypertrophy Secondary to Portal Cirrhosis. We occasionally see *very large spleens in adults* in various other conditions, such as the ague-cake spleen of chronic paludism, the enlarged spleen of portal congestion, that which rarely accompanies pernicious anæmia, the marked splenic enlargement occasionally met with in portal cirrhosis and that which is usually seen with Hanot's biliary cirrhosis. With the exception of the last, a rare condition, which is said to be associated usually with leucocytosis and which should offer no difficulty in diagnosis, these various conditions rarely present leucocytosis, and indeed leucopenia is sometimes found, especially in pernicious anæmia. The absence of leucocytosis is, therefore, useful by indicating

the simple splenic nature of such enlargements as against malignant tumor of the kidney, as exemplified in the following case: Mr. V. B., aged forty years, consulted me in May, 1900, complaining of general weakness. He was a confirmed drunkard, had thickened arteries, the edge of the liver could not be felt, and a mass with a sharp edge was felt on the left side of the abdomen reaching almost to the transverse umbilical line. The blood showed a fair degree of anæmia of a secondary type, a leucopenia of 3,120, a normal differential leucocyte count, and a normal fibrin deposit. A diagnosis was made of simple splenic hypertrophy, secondary, probably, to cirrhosis of the liver, possibly, to splenic anæmia. The patient committed suicide on January 20, 1902, affording me the opportunity of examining his organs. The liver was a typical hob-nail liver of alcoholic cirrhosis and the spleen, smaller than when previously felt (730 grams), presented the picture of simple chronic hyperplasia, secondary to cirrhosis.

The general principle which we have been considering, it must be expressly stated, applies only to adults or at least to those past the first few years of life, for the blood of infants and young children varies widely from the standard of adult life, and in childhood a marked leucocytosis is commonly found in conditions in which adults show no leucocytosis. I have dwelt at some length upon the application of this principle for the differentiation of simple enlargement of the spleen from malignant disease of the kidney, because its practical value warrants, I think, its recognition. It must not be applied indiscriminately with blind faith, but as a guide, supplementary to other methods of diagnosis, it is distinctly useful.

Many additional examples of the assistance of blood examination in diagnosis might be cited, if time permitted, but the few already given serve the purpose of concrete illustrations, as well as many. These cases speak for themselves and justify all that has been claimed for blood examination as a valuable clinical method of diagnosis in general practice. If you will employ this method you will save lives and suffering. Is it worth the trouble?

X. SYMPOSIUM ON HÆMATOLOGY

2. The Eosinophiles. Their Etiology, and Their Clinical Significance

By THOMAS R. BROWN, M. D.
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Among the factors which are tending to make medicine an exact science and to redeem it from the empiricism which has been its chief characteristic up to a comparatively recent time, nothing has played such an important part as the growth of the laboratory method of study and the application of the findings obtained therein to the intricate problems associated with the pathology and etiology of disease, the method of arriving at a correct diagnosis and prognosis, and the treatment to be employed. Within recent years, the clinical laboratory has come rapidly to the fore, and among the results obtained therein. we feel that none have been fraught with more importance than those derived from the study of the blood in health and disease.

As is well known, the blood has several constituents, red blood cells, white blood cells or leucocytes, blood plates and blood dust, as well as the plasma or fluid portion, but in this communication we shall only discuss the second of these constituents—the leucocytes—and especially that variety of leucocytes, which from the reaction of its granules is known as the eosinophile or acidophile leucocyte, its mode of origin and the significance of an increase or decrease of these cells over the number normally met with in the blood.

That the eosinophile is an important cell in the animal economy seems highly probable from the fact that it has been found in the blood of practically all mammals, as shown by the careful studies of Schwartze, Metschnikoff, Sherrington and others, and this wide distribution associated with the striking biochemical character of the cell have made it a favorite object of study among students of hæmatology. Of course, besides the general eosinophilia, that is, where there is an eosinophilic increase in the blood, there is a local eosinophilia which is extremely interesting and the study of which will undoubtedly prove of great value in determining the mode of origin of these cells. Among the places where eosinophiles are to be found in greater or less amount may be mentioned the intestinal mucosa, especially when

irritated by certain intestinal parasites and certain chemical irritants; the skin, especially in certain diseases of the skin; various tumors, especially those of a malignant nature; various exudates and excretions, especially those due to the gonococcus; nasal polyps; the secretion in bronchial asthma; and the bone-marrow, which last habitat is of special interest in connection with the enormous eosinophilic increase met with in spleno-myelogenous leukemia, and with the question of the seat and mode of origin of the eosinophiles.

In this paper we will discuss in order the proportion of the eosinophiles normally met with, the theories held regarding the origin of these cells and their functions, and then treat at some length the diseases in which an increase of these cells is met with, the significance of such an increase, and whether it is of service in diagnosis or prognosis.

As to the proportion of the eosinophiles normally met with, various observers have given slightly different figures, thus, Ehrlich gives 2 to 4 per cent., Hayem 7 per cent., Gabritschewsky 1 to 3 per cent., Müller and Rieder 1 to 4 per cent., Canon 1 to 3 per cent., Von Limbeck 2 to 8 per cent., Uskov 6 per cent., Zappert .67 to 11 per cent., and Weichselbaum 5 per cent. From our own studies of 100 or more cases of normal adults we have concluded that the average number of leucocytes per cmm. is 6,500, of which 74 per cent. should be polymorphonuclear neutrophiles, 18 to 20 per cent., small, 5 to 7 per cent. large mononuclears and transitionals, and 1 to 2 per cent. eosinophiles. Zappert regards 50 to 100 eosinophiles per cmm. as low normal, 100 to 200 as intermediate, 200 to 300 as high normal, and over 300 as pathological. According to the work of a number of investigators the percentage of eosinophiles is independent of the age of the individual and also independent of the digestion.

The question of the seat and mode of origin of the eosinophiles is obviously of great importance in determining the significance of their increase or decrease, and yet it is a question which is far from being solved at the present time. Thus, Ehrlich believes that these cells are derived from pre-existing eosinophiles often of the mononuclear variety in the bone-marrow. Uskov, Gulland, and Pfeiffer, on the other hand, believe in the non-specificity of the cell granules, that is, that all are derived from a common form, the cell appearing in each case depending on the locality and condition. Müller and Rieder believe that they are derived from the polymorphonuclear eosinophiles in the circulating blood, while various theories are held regarding a local origin of these cells; among these may be mentioned Kanthack and Hardy's theory that they are derived from fixed connective

tissue cells in the intestines and serous cavities, Howard's belief in their origin from plasma cells, various views regarding their origin from the eosinophiles normally met with in the tissues, Kischensky's view that in bronchial asthma they are formed from the neutrophiles in the mucosa, and, the theory advanced by us six years ago that in trichinosis the eosinophilic increase may be possibly due to a conversion of the neutrophiles into the eosinophiles in those portions of the muscle into which the trichinæ have wandered and set up their pathological changes.

We cannot help feeling that while Ehrlich's theory—a chemotactic origin from pre-existing eosinophile, bone-marrow cells,—probably best explains the increase of these cells in most cases; a local origin of these cells from neutrophiles is probable in certain cases, notably in bronchial asthma and trichinosis.

As to the exact nature of the eosinophilic granules many widely divergent views have been held, some founded entirely upon staining reactions, others built on a more physical basis, all more or less hypothetical, some very far-fetched and bizarre. Up to the time of the introduction of the Ehrlich stain, these coarse, highly refractive granules were regarded as fat droplets, from their original discovery in 1846 by Wharton Jones. The work of Ehrlich demonstrated that these granules are not fat, but are bodies possessing a marked affinity for the acid coloring matters, and by him and his school are regarded as a sort of nutritive reserve, built up by the cell for its own use or for excretion, and thus, according to Schwartze, somewhat resembling the aleuron granules or crystalloids of plants. Zappert and Przewoski insist that these granules are of the nature of proteid, and Pouchet and Bannwarth have regarded them as related to or identical with hemoglobin. Sherrington, on the other hand, regards them as a form of albumen, while Kanthack and Hardy believe them to be secretory products containing a zymogen, giving rise to alexines or other substances of great activity in phagocytosis and in conditions of inflammation and intoxication; and Noesske from a study of the eosinophile cells in their relation to the infectious surgical diseases and tumors, concludes that the eosinophiles play the rôle of protectors against the invasion of bacteria. In all the above views the granules are regarded as paraplastic in their nature, but Gulland holds that they are plasmatic, in other words, they represent an altered condition of the miscrosomes, and are connected by a mitoma.

Now let us discuss as briefly as possible various diseases in which eosinophilia is met with and consider the value of this increase in diagnosis and prognosis.

In leukemia the enormous increase of these cells in the spleno-

myelogenous form serves to differentiate it from the lymphatic leukemia, in which the eosinophiles are practically absent, and the presence of the eosinophilic myelocytes is of importance in showing the part the bone-marrow plays in the origin of the eosinophiles. In bronchial asthma the increase of the eosinophiles in the blood is quite marked, sometimes reaching more than thirty per cent, and this in association with the appearance of large numbers of the eosinophiles and Charcot-Leyden crystals in the sputum, serves to differentiate this form of asthma from the cardiac or renal asthma.

In connection with the association which is frequently noted between the eosinophiles and the Charcot-Leyden crystals it seems worth while for us to call attention to the work which we have done regarding the origin of these crystals. From a large series of carefully performed experiments in which the eosinophiles were exposed to various influences, we have been able to definitely show that Charcot-Leyden crystals are not direct crystallization products of the eosinophiles, and in this conclusion Müller of Vienna coincides as a result of his experiments in pemphigus.

There is no series of diseases in which eosinophilia is more interesting or more important than that due to various intestinal parasites especially the trichina spiralis and the uncinaria or anchylostoma duodenale. In 1897 we reported a case of trichinosis in which a careful study of the blood showed a marked eosinophilia associated with a high grade of leucocytosis, and the result of the study of this case and of two cases studied by us the next year led us to the following conclusions:

(1.) There is a marked increase in the percentage of the eosinophiles in the blood in trichinosis associated with a considerable leucocytosis, and this increase may be used as a diagnostic sign in this disease. (2.) That this disease in its sporadic form is more common than has hitherto been supposed, so that a systematic blood examination should be undertaken in all cases with indefinite intestinal, muscular or febrile symptoms in the hope that in some, at least, of hitherto undiagnosed cases the diagnosis may be reached.

We reported an additional case in 1899, and of these four cases the last three were diagnosed entirely by blood examination, as two of them had been previously diagnosed as typhoid fever and one as aestivo-autumnal malaria. In all four cases the diagnosis was substantiated by an excision of a small piece of muscle and the demonstration of living, active trichinæ therein. In the first case the eosinophiles reached 68.2% and the leucocytes 35,700 c.m.m. In the second case 42.8% and

13,000; in the third case 49% and 17,000, and in the fourth case 48% and 18,100. From our careful study of the blood and muscle in these cases we hold that it is highly probable that in this condition, at least, the eosinophiles are derived from the neutrophiles, transition taking place in the affected portions of muscle. Our arguments are based on the reciprocal relationship existing between neutrophiles and eosinophiles, the original neutrophilic increase being followed by a marked eosinophilic increase, the presence of a larger proportion of eosinophiles in the veins than in the arteries of the affected portions of muscle, the presence of forms which we regarded as transitional cells between neutrophiles and eosinophiles in the affected muscle, and the fact that there was a larger proportion of the eosinophiles in the veins than in the arteries of the tissues adjacent to the affected portion of muscle.

That our conclusions have been justified has been we feel definitely proven by the increasing number of cases of trichinosis diagnosed by the blood findings, for in practically no other condition is such a high percentage of the eosinophiles associated with such a marked increase of leucocytes met with. Atkinson, Blumer and Newman, Stump, Kerr, Gordinier, Cabot, and others have reported in all between twenty and thirty cases of this disease, so diagnosed, and this corroborative evidence seems to show that the blood picture may be regarded as a definite diagnostic sign in this disease.

In anchylostomiasis or uncinariasis we have again a high grade of eosinophilia, but almost always associated with little or no leucocytosis and always associated with a progressive anæmia, often reaching a very high grade and presenting many of the clinical signs of pernicious anæmia. Zapport, Leichtenstern and others have reported marked eosinophilia in this condition. The fact that a number of cases of anchylostomiasis have already been introduced into this country from our insular possessions, and the recent demonstration by Stiles of the Bureau of Animal Industry, that the disease is met with to a considerable extent in Georgia and other of the southeastern states should make us regularly examine the blood in all cases of progressive anæmia, and if eosinophilia is also present the stools should be most carefully examined in the hope of finding there the ova or the embryos of the anchylostoma.

In the case of a number of other intestinal parasites an eosinophilia has been occasionally met with, as in the case of the oxyuris ascaris, tænia, and botriocephalus and it has also been reported in the case of echinococcus and bilharzia, while recently Calvert has demonstrated a moderate leucocytosis with an increase of the

eosinophiles, reaching 22%, in one case of filariásis. Gulland and Coles have met with this same condition, and the former found that both leucocytes and eosinophiles increased during the night when the embryos appeared in the circulating blood.

Among the acute and chronic infectious diseases in which the eosinophiles may aid us in reaching the diagnosis or giving the prognosis may be mentioned scarlet fever, tuberculosis, typhoid fever, malaria, syphilis, gonorrhœa, smallpox, and occasionally in a few other of the infectious diseases.

In scarlet fever Kotschetkoff was the first to call attention to the fact that during the first and second weeks of this disease, in favorable cases, there was a distinct increase of the eosinophiles, sometimes reaching 15%, while in the fatal cases this increase was not met with; these observations have been substantiated by Zappert, Brown, and others. In the three cases which we studied of this disease, the eosinophiles varied between 6% and 12% during the three weeks. All of these cases terminated favorably.

In tuberculosis a number of observers, notably Teichmüller, have insisted that an increase of the eosinophiles in the blood and in the sputum is a favorable prognostic sign.

In typhoid fever, all observers, notably Naegli, describe an early disappearance of the eosinophiles, and the work of this observer seems to show that the early reappearance of these cells is a distinctly favorable prognostic sign, while in chronic malaria moderate eosinphilia is often met with, and may be a real help in diagnosing this condition.

In syphilis and in gonorrhœa some observers have noted a slight eosinophilia, while in the urethral secretions in the latter disease, large numbers of the eosinophiles are usually met with.

In smallpox Weil finds some eosinophilic myelocytes, while a slight eosinophilia has been described in leprosy, mumps, tonsillitis, and beriberi.

In a number of mental and nervous diseases a slight eosinophilia has been described by Zappert, Neuser, Capps and others. Recently from twelve observations on cases of chorea of varying degrees of severity we have concluded that there is regularly a slight eosinophilia varying between 5% and 9% in this disease.

In diseases of the skin an eosinophilia both local and general has often been described. Thus, it has been met with in many cases of pemphigus and chronic eczema, scleroderma, psoriasis, pellagra, lupus, urticaria, dermatitis herpetiformis, xeroderma pigmentosum, Hallopeau's dermatitis, epidermolysis bullosa hereditaria, herpes zoster, Duhring's disease and various skin affections produced by different vesicants, notably picric acid.

The eosinophiles met with in skin diseases is of especial interest, because of the fact that Ehrlich believes that the substances produced by the disintegration of epithelial cells have a marked chemotactic effect upon the eosinophiles. We have demonstrated an eosinophilia in chronic eczema, pemphigus and epidermolysis bullosa.

An eosinophilia has been noted after the use of various drugs and poisons. Thus, Zappert, Ehrlich, Lazarus, and Grawitz report a high grade of eosinophilia after the use of tuberculin, and we have recently demonstrated a leucocytosis of 30,000 with an eosinophilia of 12% after the protracted use of acetanilid.

Among the large number of diseases in which a moderate eosinophilia has been noted we would mention two cases of Addison's disease, studied by us, and many cases of gout.

Thus, a consideration of the experimental and clinical work which has been done in connection with the etiology of the eosinophiles and the clinical results obtained from the study of these cells justifies us, we believe, in reaching the following conclusions regarding the etiology of these cells and their significance.

But before giving these conclusions let us briefly recapitulate the conditions in which an increase in these cells is noted.

(1.) Spleno-myelogenous leukemia, where besides a marked increase in the ordinary or polymorphonuclear eosinophiles we find large numbers of mononuclear eosinophiles, the so-called eosinophilic-myelocytes.

(2.) In bronchial asthma associated with the presence of enormous numbers of eosinophilic cells, often mononuclear in character, and of Charcot-Leyden crystals in the bronchial secretions.

(3. In pemphigus, where an increase is frequently met with, while in the vesicles, if fresh, many eosinophiles are to be found, although in old vesicles, or those produced in this disease experimentally by chemical irritants, very few eosiniphiles are to be found, as shown by Neusser, Kriebich, and ourselves.

(4.) In various acute and chronic skin diseases, as shown by numerous observers as mentioned above, and here the extent of the eosinophilia seems to depend largely upon the extent of skin surface involved in the disease. These cases certainly favor Ehrlich's views that substances produced by the destruction and disintegration of the epithelial cells of the skin have a peculiar attraction for the eosinophiles.

(5.) In various diseases due to intestinal parasites, the most important being anchylostomiasis, where we have a marked anæmia and considerable relative eosinophilia, with no or slight leucocytosis, as a rule, and trichinosis, where the leucocytosis is very marked and the eosinophilia very intense.

(6.) The post-febrile eosinophilia following the marked diminution of the eosinophiles usually met with during the course of the fever, the notable exception to this being scarlet fever, where eosinophilia is the rule, especially in the second and third weeks, of cases terminating favorably, as shown in our three cases.

(7.) In malignant tumors, where the condition is met with, but is by no means common. It is more likely to occur in those cases where cachexia is present.

(8.) Compensating eosinophilia seen after extirpation of the spleen.

(9.) Drug eosinophilia, of which our case of acetanilid eosinophilia is a most interesting example, and in which category the eosinophilia following the administration of camphor and cantharides must be placed.

(10.) The moderate eosinophilia sometimes met with in many diseases, as gout, gonorrhœa, syphilis, malaria, rheumatism, and, we think, we may add chorea, and probably others of the functional neuroses.

The conflicting views regarding the origin and significance of these cells, and the large number of cases in which an increase, although often of but slight amount, has been met with, leaves the question of the value of eosinophilia diagnostically and prognostically in some doubt, and yet we feel warranted in claiming that in certain cases it is of great aid both in diagnosis and prognosis.

Thus, its presence seems to differentiate scarlet fever from measles, and malaria from typhoid fever, in doubtful cases, while we feel that the rapidly increasing number of cases of trichinosis diagnosed by the finding of a marked eosinophilia associated with a marked leucocytosis, is in itself of great value in differentiating this disease from atypical cases of rheumatism or typhoid fever.

Gout may be differentiated from tuberculosis where the eosinophiles are usually much decreased, while bronchial asthma may be easily distinguished from cardiac or renal asthma by the large number of eosinophiles in the blood and sputum in the former condition, and the discovery of anchylostomiasis in our Southern States makes it extremely important that new means of diagnosis should be introduced. Undoubtedly the eosinophilia with severe anæmia, almost constantly met with in anchylostomiasis, is of great aid in diagnosing this, at present, but rarely met with disease, from pernicious anæmia.

As regards prognosis, in a number of diseases the conditions of the eosinophiles furnish us real aid, as in scarlet fever, where a high count during the second and third weeks seems to be a

favorable sign; in typhoid fever, where an early reappearance of the eosinophiles (these cells disappearing in the stage of continued fever) points to a favorable prognosis; in the anæmias, where the presence of a slight eosinophilia is a good sign; in tuberculosis, where the presence of eosinophile cells in the sputum is regarded by many as favorable prognostically, and in various surgical conditions, where, again, the presence of these cells in a condition of moderate increase is regarded as a favorable sign.

XI. SYMPOSIUM ON HÆMATOLOGY

3. Degeneration of the Erythrocyte

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Mr. President and Gentlemen:

When your Business Committee invited me to take part in this discussion on hæmatology, my appreciation of the honor was blended with a keen sense of personal unfitness to venture, before such a distinguished audience as this, the discussion of the topic suggested by your committee—a topic which has been so thoroughly investigated by others that it is at present time familiar not only to the laboratory student, but also to the alert clinician. From the latter's viewpoint, however, it may not be unprofitable to consider erythrocytic degeneration, and with this aim in view I purpose to approach it, endeavoring in the following inquiry to submerge special technicalities and to minimize the discussion of mooted and theoretical points, in the hope of applying practically our present knowledge to medical diagnosis and prognosis. The various degenerative changes affecting the erythrocyte are not to be regarded as definitely characteristic of themselves, but rather as links in the chain of clinical evidence afforded by a complete blood report, which, to be of any real value, must in turn be correlated with the general semeiology of the patient in question. In passing, it may not be out of place to remark that the province of hæmatology is not primarily to furnish absolute criteria of disease, but, on the contrary, to develop data which may prove essential either to corroborate or to complete the phenomena otherwise elicited. Interpreted thus, the blood report is of great utility in every-day routine medical and surgical practice, but hæmatology divorced from familiar bedside signs of

disease in many cases leads to erroneous conclusions and to false diagnostic inferences. To neglect well-established clinical evidence in our enthusiasm for apparently incontrovertible laboratory findings not only narrows one's diagnostic acumen, but brings into disrepute such findings among those who, perchance, have failed to correlate them intelligently.

For the purpose of description, the various degenerative changes to which the erythrocyte is subject may be classified under the following headings:

I. Alterations in viscosity.
II. Simple decoloration.
III. Total necrosis and disintegration.
IV. Deformities of shape and size.
V. Atypical staining reaction.
VI. Megaloblastic forms.
VII. Granular basophilia.

It is to be understood that no hard and fast line of demarcation can be drawn between these groups, since the several changes here included are more frequently encountered in combination than singly. These changes, it will be noted, are such as can be studied by simple microscopical examination of the fresh blood and of the stained film, and their recognition involves the application of familiar and simple technical methods, easily acquired and available for practical every-day work. Of these methods it seems inadvisable to speak here in detail.

I. *Alteration in Viscosity.* When a drop of blood, collected upon the surface of a glass slide, is allowed to remain exposed to the air, the slippery, greasy feeling of the freshly shed blood is soon replaced, as coagulation progresses, by a sensation of stickiness or viscosity readily appreciable by the finger. In normal blood the erythrocytes, by virtue of their adhesiveness, tend to adhere face to face, and under the microscope are seen to cling together like a stack of coins. When this inherent property of adhesion is increased (hyperviscosity) they lose this normal tendency to form rouleaux, and accumulate in large, irregular masses, in which the distinctive histological attributes of the cells are masked or lost. The individual erythrocytes detached from such a mass may show every imaginable type of distortion, losing their typical biconcavity and disc-like form, and being converted into elongated misshapen bodies which bear not the slightest resemblance to the original cells. When this property of adhesion is greatly enfeebled or lost (hyperviscosity) the erythrocytes form neither rouleaux nor irregular masses, but float free in the plasma as isolated cellular entities. The viscosity of the whole blood is apparently influenced to a large extent by the cellular elements,

chiefly by the erythrocytes, although the viscosity of the serum must also be regarded as a determining factor of more or less importance.

Hirsch and Beck, who recently published a minute study of blood viscosity, have determined that the "viscosity value," as they term it, of human blood is about five times that of distilled water, *i. e.*, the viscosity of blood having a specific gravity ranging between 1,045 and 1,055 is expressed by the figure 5.1, in comparison with that of water, which equals 1, the temperature of both fluids being the same, 38° C. Although no close relationship can be distinguished between the degree of viscosity and the specific gravity of the blood, these experimenters have apparently proved that the lower the density of the blood the less marked its adhesiveness. The latter is exaggerated in individuals living upon a largely nitrogenous diet, and is greatly modified by starvation. Weir Mitchell has observed that hyperviscosity develops when blood is subjected to the direct action of snake venom, while Stengel has noted a similar condition resulting from contaminating fresh blood with the serum of patients suffering from chlorosis, pernicious anæmia, and leukemia. Anyone who has done much blood work is familiar with the marked fluidity of the fresh specimen in the high grade anæmias, and with the diminished viscosity of the erythrocytes, and their disinclination to form rouleaux under such circumstances.

II. *Simple Decoloration.* Under the microscope the normal erythrocyte appears of uniform color and density, save for a somewhat paler area corresponding to its central biconcavity, whence the tint insensibly blends with a relatively darker zone paralleling the periphery of the corpuscle. This evenness of color depends upon the homogenous distribution of the hemoglobin throughout the cell stroma, for, in the light of our present knowledge, we must accept Rollet's view, that the red corpuscle consists of a spongy network or stroma in which the hemoglobin is embedded, possibly in combination with a nucleo-proteid of the cell. We must decline to adopt, for obvious reasons, Schäfer's theory, that the erythrocyte is a vesicular mass, the essential parts of which are an external enveloping membrane and an internal fluid content.

Simple loss of hemoglobin and consequently decoloration of the erythrocytes is a familiar change in the blood deteriorations of a moderate grade, notably in those forms of secondary anæmia and of chlorosis which involve neither conspicuous hemoglobin and cellular deficiencies, nor deformity and nucleation of the erythrocytes. The decoloration may be quite uniform throughout the cell, beginning as a symmetrical enlargement of the normally

lighter area, and spreading progressively toward the periphery. A cell thus affected sooner or later loses its normal biconcavity, and, in the extreme instance, is converted into a practically colorless shell or phantom, which would be invisible were it not for its faintly colored marginal zone, where a small amount of hemoglobin tends to persist, in spite of its dissociation from the rest of the stroma. "Hayem's achromacytes" and "Ponfick's shadow corpuscles" are suggestive terms by which these extreme examples of cellular pallor are designated. Or, instead of this uniform decoloration, the cell may exhibit patches of pallor of unequal intensity, in case the degeneration involves several portions of the stroma simultaneously, causing more rapid and greater hemoglobin loss in certain areas than in others. Still other corpuscles show an irregular enlargement of their hyaline centers, with the ultimate production of various irregular figures, as well as streaky elongated pale areas overlying parts of the stroma from which the hemoglobin has been removed. In whatever form these hemoglobin-free areas develop, they always exhibit in the dried and fixed blood film a selective affinity for basic stains, such as methylen-blue and thionin, by which they are colored with abnormal intensity.

This phenomenon of simple decoloration has been exhaustively studied by the Italian school, especially by Maragliano and Castellino, who interpret it as a form of endoglobular degeneration or necrobiosis, and by Celli and Marchiafava, who independently have investigated the process in connection with malarial infections. It has been described also by Hayem under the caption "état cribiform," and by Klebs as a "pseudonucleation." The so-called vacuolation of the erythrocytes in the fresh blood film which has been exposed to external influences furnishes an excellent illustration of this type of cell degeneration excited artificially.

III. *Total Necrosis and Disintegration.* A general structural change, with ultimate breaking up of the erythrocyte, represents, in pathological blood, an extreme grade of degeneration, far more crippling in its effects than the process of simple decoloration, to which reference has just been made. These two changes not infrequently coexist in the same corpuscle, or they may progress independently of each other. Cell necrosis of this type is first betrayed by the development of numerous small, elevated, teat-like processes in the stroma, and sometimes by a corrugation or ridging of the flat surface of the cell. The whole corpuscle then appears to become actively ameboid, and this motility, plus its structural weakening and loss of resiliency, tends sooner or later to convert it into a misshapen body, or poikilocyte. From the

periphery of a corpuscle thus affected small bits of the stroma may be seen to extrude, break off, and float free in the plasma. These miniature vesicular masses may or may not be deeply colored, according to the amount of hemoglobin which they contain; as a rule, they show a decided affinity for basic stains, although the erythrocyte from which they are derived may not exhibit this tendency.

Decoloration and general necrosis of the erythrocytes may be studied in the fresh film of both normal and pathological blood. In normal blood they result purely from prolonged exposure of the cells to the air, the hemoglobin dissociation beginning with from one-half to one hour, and the cell destruction within from three to four hours, after the preparation of the slide. In pathological blood the changes are regarded as an evidence of an increased toxicity of the plasma, but to this principal factor of the degeneration should also be added another essential cause—the lowered resistance of the erythrocytes in consequence of which they become especially susceptible to injury even by a normal plasma, as well as by exposure to extraneous influences. In disease, therefore, these changes develop with much greater rapidity than in normal blood—in fact, they are often demonstrable immediately after the blood has been withdrawn from the body. Simple decoloration is regarded as a more favorable sign than total necrosis and fragmentation of the erythrocytes, and is generally symptomatic of more moderate grades of anæmia than those in which the latter changes prevail.

IV. *Deformities of Size and Shape.* In all anæmic states it is common to find, in association with normal erythrocytes, cells more or less increased or decreased in diameter, as well as others whose shape is variously distorted—often so strikingly that they bear but a slight resemblance to the typical discs of normal blood.

Well-defined increase in the size of an erythrocyte, without notable alteration in its contour, stamps it as a megalocyte or macrocyte. The diameter of such a cell usually ranges between twelve and fifteen micromillimeters, but in the extreme instance it may equal or exceed twenty micro-millimeters. Its stroma generally, but not invariably, is pale, owing to a relative deficiency of hemoglobin, and it shows more or less tendency to react toward a basic dye, in the stained film. There are at least two tangible views regarding the origin of the megalocyte: the first interprets the change as an actual giantism of the cell, which is bred in the bone-marrow from a correspondingly large nucleated antecedent; the second regards it as a hydropic enlargement of the cell, which, by imbibition of fluid from the surrounding plasma, swells, loses its distinctive double concavity, and becomes pale, as its hemo-

globin is relatively diminished in proportion to the extent of this cellular dropsy.

The microcyte illustrates the decrease in the size of the erythrocyte under pathological circumstances, the cell thus formed consisting of a small vesicular body, from three to five micromillimeters in diameter, and having, as a rule, its stroma colored more deeply than normal. Extremely small cells, not more than three micromillimeters in diameter, of high refraction, regularly spherical shape, and very dark color, have been dubbed "Eichhorst's corpuscles." They were once regarded, in the dark ages of hæmatology, as characteristic of primary pernicious anæmia, but are now known to carry no such clinical import. Microcytes may enter the blood stream as such, their dwarfism being directly related to a defect in hemogenesis. In other instances, and these are many, they may be simply products of corpuscular budding and fragmentation, doubtless stimulated by increased plasma toxicity plus inherent lowered resistance on the part of the cells; in consequence of these factors the erythrocyte extrudes portions of its stroma, which circulate for a time in the blood as miniature cells containing hemoglobin, and tend to assume their ancestors' biconcavity and disc-like contour. In this connection, it does not seem unreasonable to presume that the bodies known as Müller's "hemokonien" or "blood dust" may represent this phase of microcytosis.

Any erythrocyte, regardless of its size, is known as a poikilocyte when its shape distinctly deviates from normal. Such cells present an infinite variety of deformities, ranging from those showing merely a slight ovoid tendency to those distorted into the most *bizarre* figures imaginable. These irregularities have been likened to various objects, such as, for example, a gourd, a canoe, an oval disc, a sausage, a beak, a spindle, and a horseshoe. Minute rod-shaped forms, the so-called "pseudo-bacilli" of Hayem, are interesting on account of their rapid oscillations in the plasma, and their superficial resemblance to large, unstained bacilli. It is probable that poikilocytosis can generally be attributed to an increased toxicity of the plasma whereby the erythrocytes, which, it is to be presumed, are abnormally vulnerable, readily succumb to its destructive influences, and alter structurally. It is also possible that poikilocytes may arise in consequence of a defect in hemogenesis, whereby ill-shaped, as well as normal, erythrocytes enter the circulation from the marrow.

Deformities of size and shape usually are marked in relation to the severity of the anæmic process which they mirror. In some cases the changes are moderate, and tend to affect the great

majority of the cells; in others, on the contrary, it is apparent that but relatively few corpuscles are affected, but in these the distortion is prone to become most striking. The three varieties of deformity above noted are practically always associated, but in certain cases it is possible to distinguish a prevalence of one or the other type. Thus, in anæmias characterized by marked cellular loss, notably in pernicious anæmia, a general tendency toward megalocytosis is practically a constant feature of the blood report; while in those in which the poverty in erythrocytes is relatively less decided, as in chlorosis, a general tendency toward mycrocytosis is the rule. This statement is, of course, very general, and subject to exceptions in the individual case. It is a familiar clinical observation that an anæmia characterized by oversized erythrocytes is more serious and less amenable to treatment than one in which undersized corpuscles prevail.

V. *Atypical Staining Reaction.* In the stained specimen the normal erythrocyte invariably shows a strong elective affinity for a single, acid dye, by which it is colored in a characteristic manner. For example, if a solution is used which contains both an acid and a basic dye (*i. e.*, eosin and methylen-blue, or eosin and hematoxylin), the erythrocyte is stained the tint of the acid element, eosin; while with the triacid mixture, which is so formulated that an acid, basic, and so-called neutral principle may be selected by the different elements subjected to its action, the erythrocyte is always stained the color of the acid Orange G. component of the reagent. Thus, under conditions of health the red corpuscle remains consistently *monochromatophilic,* whether the technique of preparing the film involves simple single or double, or more elaborate panoptic, staining methods. Under certain pathological conditions, however, some of the erythrocytes lose their affinity for a single, acid dye, and, with mixtures of both acid and basic dyes, exhibit a definite predilection for the latter, and are therefore said to be *polychromatophilic.* Thus, in eosin and methylen-blue preparations such corpuscles are tinged violet or purple, or grayish-red, instead of the pure rose color of eosin; while with the triple stain various shades of purple, or reddish-brown, or yellow and pink flecked with deep crimson, replace the rich orange tint of the normally stained cell. Cells thus disfigured are likely to be unevenly stained, and to show contrasting light and dark areas, the latter usually being most conspicuous toward the periphery of the corpuscle, or, if the latter be an erythroblast, along the perinuclear zone of the stroma. As already pointed out, the areas of pallor in a cell undergoing simple loss of hemoglobin are especially prone to show this basic tendency, but it does not necessarily follow that polychrom-

atophilia is striking in relation to the diminution in the individual cell's hemoglobin content.

It is in the megalocyte and in its narrow antecedent, the megaloblast, that polychromatophilia is most prone to develop and to become most striking; but the change is by no means confined to cells of this variety, since it may also affect, though with much less frequency and usually less conspicuously, the various other types of degenerate erythrocytes.

The exact manner by which polychromatophilia arises is still a mooted point in hæmatology. Ehrlich, who first described the condition, under the term "anæmic degeneration," regards it as an evidence of corpuscular decay, involving a coagulation necrosis of the cell, whereby the discoplasm loses its property of combining with acid, protoplasmic dyes and displays an avidity for basic, nuclear stains. On the contrary, Gabritschewski, Askanazy, Schaumann, and others believe that the change betrays not old age, but youth of the cells, whose development has been suddenly aborted at a point of their full, normal maturity. Dissection of these opposing views and their attempted reconciliation is hardly germane to this inquiry, interesting as these different opinions may be as pathological problems. It is important, then, simply to bear in mind the facts that no normal erythrocyte ever shows polychromatophilia, and that cells which exhibit this alteration are functionally imperfect, owing either to a simple deficiency in their hemoglobin content, to morbid structural changes readily detected under the microscope, or, perhaps, to subtler inherent defects attributable to faults of development. Thus, the polychromatophilic cell may be quite normal save for its hemoglobin poverty; it may be old, deformed, and necrotic; and it may be so young that its nucleus still persists. In any event the fact remains that every cell in a stained specimen prepared with perfect technique, which shows this deviation from the normal reaction to the anilin dyes, must be considered degenerate, in so far as this term implies crippled function.

Polychromatophilia may be encountered in any of the high-grade anemias, regardless of their type, but in pernicious anemia and in leukemia this change constitutes a feature of the blood report which is to be regarded as almost constant. In chlorosis, notwithstanding the unusual hemoglobin poverty which is rather characteristic of this disease, I have seldom noted atypical staining of the erythrocytes, unless the cellular decrease was also extreme. In general terms it may be stated that polychromatophilia is a sign of extreme blood impoverishment, but by its presence alone no prediction is warranted as to the ultimate outcome of the anæmia of which it is symptomatic.

VI. *Megaloblastic Forms.* The detection of nucleated erythrocytes conforming to the megaloblastic type constitutes one of the most valuable and distinctive clues afforded by hæmatology, for cells of this class are absolutely foreign to the blood stream, as well as to the bonemarrow, in post-uterine life, and hence their presence in the stained film is conclusive proof of a severe, though not necessarily fatal, anæmia.

The megaloblast, it will be remembered, is the immediate antecedent of the megalocyte, into which it is evolved by the absorption of its nucleus (Pappenheim and Israel; Neumann and Kolliker). It is a natural inference, therefore, to presume that these two forms of cells are associated, and, clinically, this is generally found to be the case— at least, it is true that megaloblasts never exist without megalocytes, although the latter may be found alone in case they owe their origin to simple dropsical swelling rather than to nuclear loss. Megaloblasts are found in the circulating blood only in conditions involving more or less reversion of the bonemarrow to the fetal type, owing to which this tissue manufactures not only large numbers of defective giant megalocytes, but also throws into the blood stream some of their nucleated precursors of embryonal type. The discovery of megaloblasts must be regarded as a sign of striking degeneration of the hæmatopoietic organs, involving imperfect hemogenesis of embryonal character, and hence the significance of this variety of erythroblasts is diametrically opposed to that of the normoblasts. The latter, though associated with severe anæmic states, are the expression of an active and overstimulated, but normal adult type of blood manufacture, and reflect a regenerative, conservative process; they invade the blood stream when the marrow, in consequence of demands made upon it for new erythrocytes to replace those destroyed by an anæmic state, furnishes not only a quota of normal cells, but also allows the escape of an occasional immature nucleated form, or normoblast. It cannot be too forcibly emphasized that the megaloblast and the normoblast each represents a radically different type of corpuscle, the origin and clinical significance of which are entirely dissimilar.

The typical megaloblast is an unusually large round or ovoid cell, measuring about ten to twenty micro-millimeters in diameter, and provided with a relatively large-sized nucleus, which is, as a rule, situated toward the periphery of the cell body. The stroma of the megaloblast frequently appears swollen, enlarged, and marked here and there by depressed and elevated areas and perhaps by distinct flaws and cracks which fail to take the stain; generally it has a more or less regular contour, although it may be conspicuously deformed and distorted; and it is commonly poly-

chromatophilic, exhibiting combinations of color in endless variety. With the triple stain the body of the cell is usually tinted a dull tan color with deeper shadings of burnt-sienna about the nucleus and near the periphery; or a diffuse crimson tinge may predominate, as if the stain contained an excess of fuchsin. Other cells are likely to be streaked and blotched with yellow and deep brown patches, and still others stain a diffuse purple, often blending into a light pink tone in certain areas. The nucleus, which occupies the greater part of the cell body, is composed of such a delicate and scanty chromatin network that it shows but a slight affinity for basic dyes, in consequence of which it generally stains a pale sea-green or greenish-blue tint with the triple mixture. This prevailing green tint is frequently stippled with delicate dots of vivid crimson or deep purple which produce a pseudogranular effect by their contrast with the lighter undertone. In some cells the nuclear structure is coarsely mottled here and there by irregular areas of deep blue or purple. Many other equally striking tinctorial reactions to the analin dyes are also to be found in other nuclei of this type. The nucleus of the megaloblast is generally differentiated sharply from the cell body by a distinct hyaline, unstained margin which encircles it and is thrown into bold relief by the nuclear and stroma stain on either side.

As already remarked, megaloblasts occur in the circulating blood only in the severe types of anæmia which are characterized by hemogenesis approximating with greater or less fidelity that of the fetus. An occasional megaloblast may be encountered in various chronic anæmias of striking intensity, but in such conditions they are invariably outnumbered by the normoblasts with which they are associated. But in only three conditions, namely, pernicious anæmia, bothriocephalus latus infection, and nitrobenzole poisoning, have megaloblasts been found to be the predominating form of erythroblast. In thirty-seven consecutive cases of pernicious anæmia I have found, by differential counts of the erythroblasts, that the megaloblasts outnumbered the normoblasts in all but five. The average proportion of the former to the latter was somewhat more than two to one, and in seven cases only megaloblasts occurred. In contrast to this, I may cite the blood findings in twenty-six cases of leukemia (fifteen of the spleno-medullary, and eleven of the lymphatic type, respectively). In this disease the megaloblasts invariably were outnumbered by the normoblasts, which in six of the cases were the only type of nucleated erythrocyte detected, and the proportion of normoblasts to megaloblasts in these twenty-six cases averaged about four to one. Comparing the numerical estimates of both

forms of erythroblasts in these two forms of primary anæmia, it was determined that the average estimate in pernicious anæmia was 220, the highest 924, and the lowest 3, cells per cubic millimeter of blood; while in leukemia the average was 5,800, the highest 12,913, and the lowest 748 to the cubic millimeter. These figures illustrate the striking abundance of erythroblasts in leukemia, and also show that in this disease the compensatory hemogenesis does not counterfeit the embryonal type so closely as it does in pernicious anæmia. From a rather close study of the question, I am convinced that megaloblasts are the prevailing form of nucleated red cells in every genuine case of pernicious anæmia during its active stages. In the exceptional case their absence may be explained by the examiner's carelessness, or, perhaps, by the fact that the marrow has become so wrecked by the specific toxines of the disease that it is no longer capable of turning out even these makeshifts of corpuscles. It is also true that during the periods of remission, the megaloblastic phase of the blood picture, as well as its other characteristics, temporarily disappears. The presence of megaloblasts in leukemia betrays the existence of fetal marrow changes, and their association here with such an amazing profusion of normoblasts indicates, furthermore, that active blood regeneration accompanies this degenerative lesion.

The megaloblastic blood picture of bothriocephalus anæmia, described by Askanazy, may be explained by assuming that in this infection certain unknown poisons, elaborated by the parasite, are absorbed by the human host, in whom they provoke fetal marrow changes. After expulsion of the worm the megaloblasts rapidly disappear from the blood, and are replaced by normoblasts, which in turn disappear, as the patient's blood becomes normal and his health restored. In nitrobenzol poisoning, Ehrlich and Lindenthal have observed a similar predominance of megaloblasts, and in this condition it is also to be assumed that a similar embryonal degeneration of the marrow must have been excited by the intoxication.

VII. *Granular Basophilia.* In many of the anæmias the film stained with methylen-blue, thionin, or hematoxylin shows a peculiar basic granular condition of the stroma of some of the erythrocytes, to which the term granular basophilia and granular, punctate, or basic degeneration are applicable. In the specimen stained, for example, with eosin and methylen-blue these granules appear as a fine or coarse blue stippling, or, rarely, as blue linear markings, or as a combination of these three phases. These basic areas are either distributed quite uniformly through the stroma, or are grouped in a more or less compact mass toward the margin

of the cell body, against the rose tint of which their intense blue-color shows conspicuously. Both normally shaped, distorted, nucleated, and non-nucleated corpuscles may be in this manner basically stippled, and it is not uncommon to find that these cells also bear evidences of polychromatophilia.

The exact nature of these granules has provoked a great diversity of opinion among different investigators, whose differences may be narrowed down to these two prevailing views: first, that they are the product of nuclear fragmentation, and; second, that they represent a true stroma degeneration. Among those who urge the acceptance of the first view may be mentioned the names of Askanazy, Litten, Lararus, and Engle; while Grawitz, Hamel, Moritz, and Stengel are among those who have advanced not only clear arguments, but undeniable proofs that the process is in no wise of nuclear origin, but that, on the contrary, it is essentially protoplasmic. In order to get at the truth of this debated topic, it is necessary here to refer briefly to the chief phases of these opposed theories and to the facts by which they are substantiated.

First, to inquire if the granules are nuclear debris or products of fragmentation, occurring as the result of a nuclear absorption by which the erythroblast ultimately becomes a non-nucleated erythrocyte, or normocyte. To support this belief, it is reasonable to presume that some of the cells should exhibit changes which may be interpreted as transitional stages between the typical erythroblast and the non-nucleated granular erythrocyte. But, from the evidence thus far presented, it cannot be said that such forms have been conclusively demonstrated, although both Litten and Ewing appear convinced that they exist. The former has minutely described them, both in the circulating blood and in the bone-marrow of a single case of pernicious anæmia; while the latter states that, in this same disease, he has observed the transitional stages and fine subdivision of megaloblastic nuclei. These, however, are isolated annotations, and lack corroboration by the many other investigators who have studied the question. Personally, it has never been my good fortune to discover such intermediate forms of cells, although both in pernicious anæmia and in leukemia, in which erythroblasts and granular erythrocytes commonly coexist, I have tried to distinguish the progressive pallor and fragmentation of the nucleus with coincident multiplication of the granules, such as Litten describes, and thus to become convinced of the truth of this tempting hypothesis. Stengel, White, and Pepper have recently offered most positive evidence against the nuclear origin of the granules, by demonstrating in granular erythroblasts signs of active karyomitosis—a finding that seems conclusive, since it is hard to believe that a

nucleus can simultaneously divide, by mitosis, and fragment, by rhexis. These workers also found precisely similar types of nucleated and granular cells in the bone marrow and blood, whereas, according to the theory of nuclear fragmentation, the marrow naturally should contain numerous examples of transitional forms, for in this tissue the transition of erythroblasts to erythrocytes occurs normally.

To support the second view, that granular basophilia betrays a form of stroma degeneration, presumably specific, it is necessary only to refer to the clinical and experimental work of Grawitz and his assistant Hamel, and to the careful investigations recently completed by Stengel and his associates.

Four years ago Grawitz declared that basophilia of the erythrocytes was a specific degenerative change, and that the exciting cause was possibly some form of blood poison which acted deleteriously on the circulating cells. His chief conclusions may be briefly summarized as follows:

(*a*) Granular basophilia may be demonstrated in peripheral blood which shows absolutely no other signs of degeneration.

(*b*) Transitional stages between typical erythroblasts and granular erythrocytes exist neither in the circulating blood nor in the bonemarrow.

(*c*) Erythroblasts with typically intact nuclei may show striking basophilia of the cell body.

(*d*) Experimentally, granular basophilia of the circulating erythrocytes can be promptly excited in animals by the administration of various toxic substances, as well as by subjecting them continuously to high degrees of heat. After the animal's recovery from either of these harmful influences, the granules disappear from the blood.

Stengel and his pupils have, step by step, confirmed on the whole these data, and, in addition, have recorded the very important observation already referred to, namely, that basophilic granules are not incompatible with nuclear mitosis in the very cells whose stroma they occupy—an obviously absurd combination of progressive and retrogressive changes. These workers also lay stress upon the absence of transitional forms of cells in leukemic blood, in which erythroblasts showing various signs of nuclear degeneration (such as atrophy, pyknosis, karyolysis, and karyorrhexis) are not uncommonly demonstrable. They were, however, unable to determine the exact seat of origin of the degeneration, whether it resided in the peripheral blood, in the portal circulation, or in the bone-marrow.

Whatever be its mode of production, the fact remains that granular basophilia of the erythrocytes is a change never found

in the blood of the normal individual. It is rational to regard it as a stigma of cellular degeneration, and its detection is sufficient grounds for directing the clinical inquiry toward a search for an adequate underlying factor of some anæmic condition. For convenience's sake, granular basophilia may be discussed under two headings: experimental and clinical.

Experimental basophilia, as Hamel first proved, can be excited in animals by the administration of small doses of lead acetate, either by the mouth, or by subcutaneous and intraperitoneal injection. The minimum amount of the metal requisite to cause this change has been found to be 0.03 gramme, although most experimenters have employed larger amounts, ranging as high as 1 gramme. Later experiments, notably those of Moritz, Behrendt, Bourret, and Stengel, have fully confirmed the truth of this observation. It appears that, under the effect of lead, the erythrocytes become faintly basic within so short a period as twenty-four hours after the initial dose, and that the granules become strikingly developed by the end of three days, if the dosage is continued. (White and Pepper). When this treatment is discontinued the granules disappear after the lapse of a variable length of time, which varies according to the amount of the lead salt administered, the gravity of the toxemia, and the susceptibility of the animal.

Grawitz succeeded in producing granular basophilia in white mice kept exposed continuously to a temperature ranging between 37° C. and 43° C., the change first becoming evident after eight days, and, as the animals later became accustomed to their new environment, quite disappearing in course of time. In these experiments it was observed that the development of the basophilia was associated with a distinct dilution of the blood, and with an obvious decline in the animals' health, and that as the granules disappeared, and the blood again became normal and the animals apparently thrived, some of them remaining alive and well for months, in spite of their confinement and exposure to excessive heat. The results of these experiments have a certain clinical bearing, in that they tend to show that possibly some of the tropical anæmias may be due, at least in part, simply to the effects of sustained high temperature, which in the newcomer to the tropics is prone first to excite an anæmia, which later may disappear, as the individual becomes better adapted to the new surroundings and mode of life.

Grawitz has also succeeded in exciting at will distinct granular basophilia by the administration of various proprietary preparations of hemoglobin devised as substitutes for the different iron preparations in the treatment of anæmia. The exhibition of such

drugs in ordinary dosage was promptly followed by a basophilia, which was aggravated by increasing the dose, and which soon disappeared after the medicament was discontinued. These results were observed both in anæmic and in healthy individuals, but the basophilia developed much more rapidly and more strikingly in the former. From these observations, it appears that the presence of blood in the gastro-intestinal canal is attended by the elaboration of certain unknown toxic substances, possibly evolved by the action of the intestinal bacteria of decomposition upon the hemoglobin preparation ingested, and that the absorption of these noxious principles acts deleteriously on the blood cells of the individual. This action Grawitz terms "plasmotropic," or one which causes the cellular defects in the bone-marrow, the liver, and the spleen, but which does not affect the erythrocytes in the circulating blood. In direct contrast to this form of blood destruction stands the process known as a "plasmolytic" action, or one by which the erythrocytes are destroyed in the blood stream, with the consequent supervention of hemoglobinemia.

Among the noxious substances, by the administration of which it is possible to excite granular basophilia, are tin chlorid (Löwenthal), copper (Sabrazes, Léger, Bourret), pyrodin and atropin (Strauss), methylen-blue and toluilendiamin (Bloch), and phenylhydrazin (Kaminer and Rohnstein).

Clinically, granular basophilia is encountered in a great variety of anæmic conditions, chief among which are lead poisoning, pernicious anæmia, leukemia, malarial fever, intestinal helminthiasis, sepsis, and malignant disease. In addition to these conditions, many of the secondary anæmias, resulting from other factors, also are associated with this change.

Lead poisoning, be it symptomless or frankly developed, is the one condition in which granular basophilia is constantly found. In fact, it is possible to demonstrate this sign in lead workers and painters who show absolutely no other manifestations, either subjective or objective, of saturnism. In such cases the erythrocytes are studded with these granules long before other blood changes supervene, and before the patient's gums become discolored, his wrists powerless, his bowels constipated, and his headache and abdominal colic annoying. Moritz first demonstrated this fact in a series of six cases, and later Hamel obtained similar results in twenty-five. White and Pepper examined twenty-one lead-workers, none of whom had symptoms, and found the granules in every case, about one-half of which also showed moderate corpuscular anomalies, chiefly defects of size and shape, and a few, atypical staining and normoblastic nucleation of the cells.

Simon found granules in twenty lead-workers, but two of whom had active symptoms.

The fact that pallor, without decrease in the number of erythrocytes and loss of hemoglobin, is a familiar objective symptom in many lead-workers, formerly was regarded as a proof that this class of artisans were only apparently anæmic, the source of the deception being referred to a general vaso-motor constriction thought to be due to the effect of the metal. The presence of basophilia in such instances, however, dissipates this error, for it shows that, although perhaps there is no notable deficiency in the number of erythrocytes and their hemoglobin content, the cells are, in reality, distinctly degenerate. As the systemic effects of the lead toxemia disappear under treatment, the granules also disappear, usually in from four to six weeks after proper elimination has been instituted. While the presence of granular basophilia can in no sense be interpreted as a conclusive sign of plumbism, it is of symptomatic value in doubtful cases. As a negative piece of evidence, too, it is of utility, for the absence of basophilia in an individual whose symptoms suggest saturnism is a fairly reliable indication that this intoxication is simulated by some other lesion, such, for example, as a gastric neurosis or an appendiceal colic.

In *pernicious anæmia* basophilia of the erythrocytes is practically a constant blood change in every well-defined case. It has been uniformly present in the last six consecutive cases of Biermer's disease which I have had occasion to examine with this purpose in view. But, although this disease illustrates primary anæmia in its most intense form, it cannot be said that the abundance of the granules corresponds definitely to the degree of the blood impoverishment; it can, however, be positively asserted that they become less numerous, and may even disappear, during the periods of temporary improvement in the blood and in the patient's general health, only to reappear as the inevitable relapses occur. Under these circumstances the behavior of the granules corresponds to that of the nucleated, deformed, and atypically colored erythrocytes, and, with them, it constitutes a rational means of determining the progress of the disease.

In both forms of *leukemia* the granules are also found with great constancy, but they do not seem to be so numerous as one would be led to expect, judging from the other striking blood changes which accompany this type of anæmia. From a limited number of cases at my disposal, I have been unable to determine whether or not any relation exists in leukemia between the abundance of the granules, the height of the leukocyte count, and the behavior, quantitatively and qualitatively, of the erythrocytes.

In the late, anæmic stage of Hodgkin's disease, the granules appear in the majority of instances, but they are not to be detected early in the disease, when the blood shows little or no deviation from normal.

The *malarial fevers* involve erythrocytic basophilia in the great majority of cases, especially in those in which the cachexia is marked and the anæmia of high grade. In our service at the Jefferson Hospital, where many cases of tertian fever and an occasional estivo-autumnal infection are treated, we have come to consider basic granulation almost as familiar a blood finding as the presence of the parasites themselves. Corpuscles containing small parasites may also show the granules, and the latter may be found in the uninfected erythrocytes. Plehn, who was the first to call attention to the presence of these granules in malarial fever, assumed that they resulted from the disintegration of the corpuscles by the parasites, but, in addition to this direct action of the latter upon their cellular hosts, the change must also develop in consequence of the deleterious effects of the circulating malarial toxins, and of the secondary anæmia provoked by the infection itself.

Certain *intestinal parasites* are capable of causing not only profound diminution in the number and hemoglobin value of the erythrocytes, but also striking granular defects in their stroma. Both Schaumann and Jawein have described these combined changes in the severe anæmia secondary to *bothriocephalus latus* infection, while Grawitz's studies tend to show that basophilic granules are a prominent feature of the blood picture in the anæmia of ankylostomiasis, or, more properly, uncinariasis. Their occurrence in this form of helminthiasis he attributes to an autointoxication due to the absorption of a toxic substance developing within the patient's intestine in the blood there collected by the constant capillary oozing provoked by the hook-worm. In this instance another illustration of a so-called plasmotropic action of an absorbed toxin is also afforded. According to this theory (which as yet lacks substantiation) the toxic material presumably elaborated by the parasite, appears to be of distinctly secondary importance as a factor of the blood changes in this infection.

In *septic conditions,* whatever be their origin, the erythrocytes are prone to show granular basophilia, but with great inconstancy, according to the reports at present available. It is possible that this change develops in direct relation to the deleterious effect, in the individual case, of the septic poisons, which are known to produce, in grave infections, the rapid onset of an anæmia of the most intense degree, as well as a marked leukocytosis. Similarly, in *carcinoma* the presence of basophilia is

thought to depend upon the toxic effect of the products absorbed from the growth. In this disease it appears that the change occurs only in those cases in which the neoplasm is situated so as to allow free absorption—Grawitz, for example, frequently found granular basophilia in gastric and intestinal growths, but failed to detect it in uterine cancer, even in advanced stages. But in cancer of the stomach and intestines, if they are associated with hemorrhage, another determining factor is naturally suggested, namely, the effect upon the circulating erythrocytes of the noxious substances absorbed at the seat of the bleeding. In this connection it may also be noted that the ingestion of blood, as in the case of phthisis with pulmonary hemorrhage, may account for the presence of basophilic erythrocytes, and that liver cirrhosis, peptic ulcer, and other lesions associated with hemorrhage into the gastro-intestinal tract must also be regarded as factors of this pathological finding.

In *chlorosis* granular basophilia is extremely inconstant, and the determining factor of the change in this disease remains obscure. Judging from my rather limited acquaintance with this phase of basophilia, I must agree with the generally voiced opinion that the erythrocytes in chlorosis but rarely show this degenerative ear-mark, in spite of the striking hemoglobin deficiency, as well as of the structural alterations which they sometimes exhibit. Stengel, on the contrary, found that the granule cells were abundant in eleven of the eighteen chlorotics whom he examined, although only in a single instance were the granules themselves numerous; in all of the eleven polychromatophilia was also demonstrable, but it stood in no definite parallelism to the degree of coexisting basophilia. May it not be that the latter, when it does develop in chlorosis, may be traced to the effects of intestinal autointoxication.

Pure *tuberculosis* does not appear to produce basophilia, but if a septic infection be engrafted upon a tuberculous process, the change is prone to supervene. Among the many conditions in which basophilia does not tend to occur, diabetes mellitus, syphilis, influenza, and measles may be mentioned as prominent examples.

In conclusion, an analysis of the foregoing remarks, stripped for clinical application, appears to justify these deductions:—

I. The viscosity of the erythrocytes is influenced by cellular and plasma alterations, the nature of which is not understood. The viscosity is exaggerated by the direct agency of various toxic agencies, and diminished in anæmic states. This phase of cellular pathology is interesting from an experimental viewpoint, rather than as a finding of clinical application.

II. Simple decoloration illustrates the earliest retrograde change affecting the erythrocytes, and its intensity generally corresponds to the severity of the anæmic process by which it is excited. The change may exist alone, as in the milder forms of anæmia; or it may be combined with graver necrotic degeneration of the cells, in anæmias of greater severity.

III. Deformities of shape and size are common to all pathological blood, the degree to which such changes develop being related to the intensity of the blood impoverishment. Megalocytosis is a more serious sign than microcytosis.

IV. Atypical staining of the erythrocytes betrays an impairment of function, and, as a rule, is found most commonly in corpuscles whose hemoglobin content is subnormal. It is most striking in anæmias of the primary type.

V. The prevalence of megaloblasts indicates a fetal reversion of the bone marrow, and stamps the blood changes as pernicious, except in the anæmias symptomatic of *bothriocephalus latus* infection and of nitro-benzol poisoning. The presence of megaloblasts indicates a severe anæmia, but not necessarily one of fatal outcome.

VI. Granular basophilia, whatever may be its exact origin, should be interpreted as a sign of degeneration. It is a constant blood finding in but a single condition—lead poisoning—but is associated with many diseases involving a variable degree of blood deterioration. The experimental basophilia excited by the administration of preparations of hemoglobin should prompt an inquiry as to the wisdom of using such medicaments as substitutes for iron in the treatment of anæmia.

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XII. SYMPOSIUM ON HÆMATOLOGY

4. The Iodine Reaction in the Blood and Its Diagnostic Value

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Ehrlich,[1] in 1882 first called attention to the presence of a reaction in pus when treated with a solution of iodine.[2] Since that time various investigators have studied with considerable thoroughness the same reaction in the cells of the blood, both in health and disease. The results of these studies are reviewed, and the microscopical appearances of the reaction fully described in a recent article by Dr. R. C. Cabot and the writer.[3] In this paper it is my purpose to describe the exact character of this test, and to speak somewhat briefly of its clinical significance.

The method of preparation and examination is very simple. A thin film of blood is spread upon a cover glass (or slide) and allowed to dry in the air, no further fixation of any kind being necessary. This unstained smear is mounted on a slide with a drop of the iodine solution, and is then ready for examination. The best result is obtained by using an excess of the mixture, that all cells may come freely in contact with it, and then to squeeze out the excess by gentle pressure on the cover glass, lest the yellow of the fluid be too deep to contrast with that of the cells. The reaction takes place instantaneously, so that the specimen can be examined at once. On the other hand, if inconvenient to look for the reaction at once, the dried smears can be kept

indefinitely, as the power to react to iodine is but little changed even after several months. The preparation once made, though in the strict sense not a permanent one as is the case with those stained and mounted in balsam, can be kept for a considerable time.

The examination should be made with an oil immersion lens, and with a strong illumination of the field, preferably by daylight, as the contrast between the brown coloration or granules and the diffuse yellow of the field is much more marked.

If a slide of normal blood thus prepared be examined, all the cells will be found to have taken on a uniform bright yellow color, the nuclei of the leucocytes standing out quite distinctly as somewhat lighter and more refractile than the surrounding protoplasm. The characteristic markings of the various types of cells are sufficiently brought out readily to differentiate them. Such is the picture in normal blood. In the blood under certain pathological conditions, however, the picture is considerably changed by the presence of small or large brownish granules in the protoplasm of the polynuclear leucocytes, or a diffuse brownish tint to the same cells and by large and small brown masses outside the corpuscles. This is the "Iodine Reaction."

As suggested by this description, the reaction is of two general types, namely: (I) The extra-cellular and (2) The intracellular.

1. The extra-cellular reaction: The masses found outside the cells are usually round or oval, varying in size from a mere dot to even five or six mikrons in diameter, and though sometimes free are usually within the fragments of protoplasm so commonly seen in the blood. Their color is copper red and contrasts sharply with the uniform yellow of the field. I have repeatedly compared specimens of blood stained by Wright's method,[4] and others mounted in the iodine solution, and am convinced of the identity of these fragments of protoplasm with the blood plate.

2. Intra-cellular reaction: Three varieties are found: (a) A diffuse yellowish-brown coloration of the protoplasm of varying intensity; (b) distinct brownish granules scattered diffusely throughout the protoplasm, and (c) very large, irregular areas of a faded brown color about the periphery and frequently protruding from the body of the cell as though oozing out.[5] This last mentioned form I have seen only in the leucocytes which give evidence of extreme degeneration. In the vast majority of cases the neutrophiles alone show the reaction, though in a few instances I have seen it in the lymphocytes and myelocytes. In these cells the arrangement of the granules is entirely changed, for instead of being scattered diffusely throughout the proto-

plasm they exist as a more or less definite row of ten to thirty large, irregular areas about the very periphery of the cell.

Extra-cellular granules are present in the blood under conditions both normal and abnormal, though in health usually in such small numbers as to be found only with difficulty. Their increase has been noted under a great variety of conditions, but is so variable that it seems to be of little value except when associated with a positive intra-cellular coloration. The intra-cellular reaction is never found in health.

The variability in the number of leucocytes with iodophilic granules, as well as in the intensity, must always be borne in mind, since the intensity of the reaction in individual cells seems to be of as much importance as the number of cells affected. Very often the majority of the leucocytes in the preparation give a definite reaction; on the other hand, not infrequently only two or three per cent. are positive to iodine. It is then obviously necessary to look over a large number of cells before pronouncing the test negative. In all doubtful cases, I count at least one hundred white cells, and, if in that number none are observed with either a brown tinted protoplasm or brown granules, I consider the reaction negative.

Having described the reaction, let us now turn to a brief consideration of some of the more common diseases in the diagnosis of which a positive or negative iodophilia may be of value.

Septicæmia. In all of my series of cases of this affection there has been a very marked reaction, in most instances from one-half to three-fourths of all the neutrophiles being colored or with granules. I have noted especially in this condition the third form of the intra-cellular type described above, namely, the very large faint yellow neutrophiles with the large pale masses about the periphery. In two of my series in which there were no characteristic symptoms, and the leucocyte count was normal, the marked iodophilia aided greatly in diagnosis.

Abscess and Local Sepsis (Non-tubercular). My results in this condition correspond very closely with those of Goldberger and Weiss,[6] who conclude that the reaction is present in all advancing suppurative processes, and that its intensity is proportional to the rapidity of the purulent accumulation. The size of the abscess in itself seems to have but little effect upon the reaction, that is, a very small acute abscess may give a more striking iodophilia than a chronic one of enormous size, clearly suggesting the dependence upon the toxæmia rather than the size of the pus cavity. After free drainage of a cavity the reaction rapidly disappears, in my cases noticeably diminishing after eight to twenty-four hours. Any re-accumulation of pus is at once shown in the

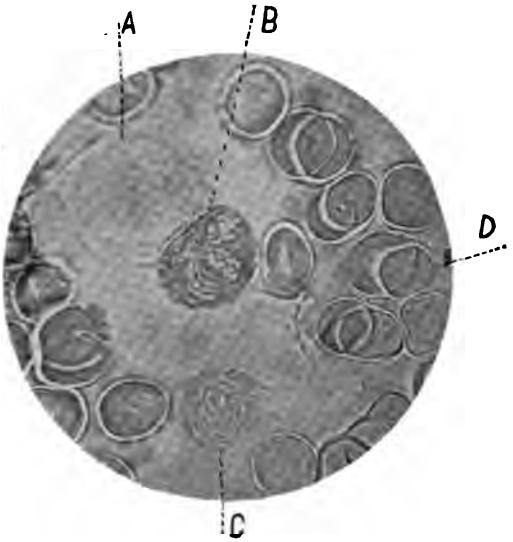

FIG. I. (X 1000)
A. Normal neutrophile. B. Neutrophile with very marked reaction. C. Normal lymphocyte. D. Erythrocytes.

FIG. II. (X 1000)
A. Normal neutrophile. B. Neutrophile with large pale areas about periphery.

FIG. III. (X 1750)
A. Neutrophile with a very marked reaction.

FIG. IV. (X 1750)
A. Normoblast. B. Neutrophile with very striking reaction.

reappearance of the reaction. In chronic suppurative processes with free drainage, even if the daily discharge is profuse the test is often wanting. Moderate local inflammation without pus if not accompanied by constitutional symptoms usually fails to give the reaction.

Appendicitis and General Peritonitis. My studies in these conditions comprise sixty-two cases. From the findings at operation I was enabled to divide them into four groups, as follows:

1. Interval appendicitis.
2. Acute appendicitis without general peritonitis or local abscess.
3. Appendicitis with local abscess, but without general peritonitis.
4. General peritonitis.

The results were striking. Under the first group were included these cases which showed no symptoms at the time of the blood examination and which at operation were found free from active inflammatory processes about the appendix. The leucocyte counts were from 7,000 to 12,000. The intra-cellular iodophilia was in every case negative, though in several the extracellular masses were somewhat increased. The cases which at operation were found with local inflammation, but not sufficiently advanced to develop local abscess or general infection of the peritoneal cavity, were included in the second groups. Here the leucocytosis ranged from 12,000 to 24,000, and the duration of symptoms from eight hours to four days. In one case of twelve hours' duration with 12,000 leucocytes and an appendix which was only very moderately inflamed, the reaction was negative. In the other twelve cases the intra-cellular reaction was uniformly positive, but usually only "faint" or "fair" in character.

The twenty cases included in the third group were uniformly of a more advanced type; all had progressed to the stage of local abscess formation, but without a definite general peritonitis. The leucocytosis, with two notable exceptions (7,400 and 8,000) ranged from 10,000 to 36,000. All cases in the group gave a definite reaction, in 50% noted as "marked."

In the fourth group were comprised those cases in which the peritoneal cavity was unquestionably involved. Here again the leucocytosis was normal (4,800 and 8,000) in two cases, both with the abdominal cavity containing free pus. With these exceptions the white counts ranged from 12,000 to 52,000. The reaction was invariably positive and on the whole more marked than in the preceding groups.

Pneumonia. Lobar pneumonia is always accompanied by a positive reaction usually intense and appearing very early, often before the signs of consolidation. In eleven of my series of cases, I examined the blood at brief intervals following the crisis and found the intensity of the reaction to diminish after six to eight hours, and usually in from twenty-four to thirty-six hours to entirely disappear. In delayed resolution, the fading of the reaction was also delayed.

Empyema. This affection always gives a pronounced iodophilia.

Pleurisy. In my series of sixteen cases, including both dry and serous pleurisy though frequently accompanied by a leucocytosis of from 13,000 to 19,000, the results were negative. In several with very marked symptoms the picture was typical of empyema, and the iodine test alone served to make the diagnosis clear.

Salpingitis. In this disease the results were less uniform than in appendicitis, though always present in all cases with a severe inflammatory process.

Gonorrheal Arthritis. I have examined but three cases, all in the acute stage, and in all found the leucocytes to react moderately.

Articular Rheumatism. In contrast with gonorrheal arthritis, uncomplicated articular rheumatism does not give a positive reaction. One case with endocarditis, pericarditis, and temperature of 104°, was positive. Livierato,[8] in this series, reached similar conclusions.

Anæmia. Chlorosis never gives a reaction. Pernicious and secondary anæmia usually yield a very moderate reaction in the late stages, but never the marked type of septic conditions. The same may be said of cardiac and malignant disease.

Acute Intestinal Obstruction. In only three instances have I found the test positive, and in these the intestine was gangrenous.

Diabetes Mellitus. Except in coma and gangrene, the intracellular reaction is absent, while the extra-cellular is invariably increased. Livierato and Kaminer report similar results.

Malaria. Of forty-nine cases, twenty-one reacted, in a few instances strikingly. I have never seen any evidence that its appearance bears any relation to the type of malaria or time of chill.

Nephritis. In very severe cases, especially uræmia, a fair reaction often occurs.

Typhoid Fever. A positive test was present in seventeen of my forty-two cases. Various complications in many served to

explain its occurrence, in the remainder it seemed to be due to toxæmia. Phlebitis, unaccompanied by gangrene, in my experience has failed to produce a reaction.

Hernia. As in acute intestinal obstruction, the results are uniformly negative, unless there is strangulation with necrotic changes.

Tuberculosis. Except in the very latest stages, I have never seen iodophilia present in tuberculosis free from secondary infection.

Let us now consider the interpretation of iodophilia and the conclusion which can be drawn from its presence or absence.

The exact nature of the substance within the leucocytes giving the color reaction to iodine has not been definitely determined, though Ehrlich, Gabritschefsky, and others, believe it to be glycogen. Whatever the substance may be, its presence seems clearly due to some degenerative process in the white cells, as evidenced by the clinical facts and microscopical appearances. Various explanations have, from time to time, been offered for these degenerative changes, but the simplest, it seems to me, is found in a toxæmia in all cases, whether of bacterial or non-bacterial origin. Such an hypothesis readily explains the striking reaction invariably found in those cases of severe sepsis, in which a leucocytosis is absent as noted above. When in the presence of a marked degree of sepsis, the blood can no longer react by reason of the overwhelming toxæmia, and a normal or hypoleucocytosis occurs, the iodine reaction becomes more intense in consequence of the resulting degeneration of the cells. This interpretation is further born out by the prompt fading of the brown granules following the free drainage of pus and by its as prompt return if the pus again accumulates. If then this reaction means a definite degenerative change in the white cells consequent upon a toxæmia, it certainly seems fair to accept it as a rough measure of the severity of a given affection. My experience with it in clinical work has furnished ample justification for such a position. I have never seen a patient with a positive iodophilia whose symptoms were not severe. While in many instances telling us nothing which we may not gain from other observations as of temperature and leucocytosis, yet it is by no means identical with them.

As is true of most laboratory tests, iodophilia has no general application in diagnosis neither is it pathogomonic of any particular disease. The information which it furnishes us is the presence of a certain condition, *i. e.*, a toxæmia, which may be produced by a great variety of diseases. It is of value only when considered as a part of the symptom complex, and only then are

any general deductions possible. The mere presence of the intra-cellular glycogen granules in a given case is consistent with a considerable variety of affections, but if considered with a group of signs and symptoms suggesting a definite condition, its significance becomes much more specific.

As will be readily seen, the reaction has both a positive and negative value in diagnosis. In my experience, including nearly a thousand examinations, it is uniformly present in the following diseases; septicæmia, abscesses, and local sepsis, except in the very earliest stages, appendicitis accompanied by abscess formation or peritonitis, general peritonitis, empyema, pneumonia, pyonephrosis, salpingitis with severe inflammation or abscess formation, tonsilitis, gonorrheal arthritis and hernia or acute intestinal obstruction if the bowel has become gangrenous. Hence, in its absence all these can be ruled out, or, in other words, no septic condition can exist without a positive reaction. On the other hand, in certain diseases the results are as uniformly negative, among them dry and serous pleurisy, simple bronchitis, articular rheumatism, uncomplicated tuberculosis, hernia and acute intestinal obstruction previous to the occurrence of tissue necrosis in the gut, and many others. In the occasional cases of typhoid or malaria with a positive reaction, no confusion should arise, for the diagnosis is usually possible by other means. In typhoid it rarely appears early.

The reaction in nephritis, cardiac and malignant disease, anæmia and diabetes is, ordinarily, not of the marked type seen in sepsis, and appears only in the late stages of the disease long after the true diagnosis is unmistakeable.

Aside from the especial value of this reaction in certain types of diseases, I have found the method a most convenient one for making a hasty general blood examination. After a little practice, one is able to judge with surprising accuracy of the number of leucocytes present, while any considerable change in the percentages of the various types of white cells is readily seen. Changes in the size and shape of the erythrocytes and presence of nucleated red cells, while less strikingly shown than in a smear stained by Ehrlich's or Wright's method, is yet sufficiently evident to be at once noted if marked; as in fresh specimens the malarial parasites are readily recognized.

One or two actual cases in which I have found the test useful may serve to illustrate its place in diagnosis.

Several months ago I was asked to examine the blood from a case which Dr. J. M. Jackson had seen in consultation. The patient was a boy of six, who had been sick for three days, though in bed only for six hours. The symptoms were rather indefinite,

but pointed strongly to appendicitis. The physical examination showed an abdomen somewhat distended, very slightly rigid with some general tenderness which was most marked in the right iliac fossa, and uncertain changing dulness in the flanks. The child seemed very sick, and the diagnosis most probable was appendicitis with a question of general peritonitis. A white count of 30,000 with a very marked iodine reaction served to confirm the diagnosis and to make the involvement of the peritoneum more probable. Operation one hour later revealed a gangrenous appendix with general peritonitis.

December 31, 1901, a man of sixty entered the Massachusetts General Hospital, giving a history of strangulated inguinal hernia of thirty-six hours' duration. The temperature was normal, the white count 15,000. Though the correct diagnosis was made, yet the seriousness of the patient's condition was not fully appreciated until a very marked iodine reaction served to emphasize it. An immediate operation showed an extensive necrosis of the small intestine, and, in spite of his seeming good condition, the patient died in a few hours.

1. Zeitschrift fur klinische Medicin, 1888, Bd. VI.
2. Iodine I grams. KI 3 grams. Water 100 c. c. Gum arabic 50 grams.
3. The Journal of Medical Research, 1902, VII, No. I, 25-42.
4. The Journal of Medical Research, January, 1902, VII, No. I.
5. See Hectoa Prog. Med. 1901.
6. Weiner klinische Wochenschrift, 1897.
7. For tables see author's article, Boston Medical and Surgical Journal, 1902, CXLVII, No. II, 289-296.
8. Deutsche archiv. fur klinische Medicin, 1894, Bd. 53.

XIII. OBSERVATIONS ON AMERICAN CLIMATES AND LOCALITIES IN THE TREATMENT OF PULMONARY TUBERCULOSIS

By JAMES K. CROOK, A. M., M. D.
NEW YORK

There is reason to believe that the scourge of pulmonary tuberculosis will eventually be mastered by the science of man. Being a specific infectious disease, the nature and causes of which are adequately understood, it requires no great strain of the imagination to foresee a time not many generations hence when this most virulent enemy to human life, will to a great extent, be eliminated as an important factor in mortality statistics. It is well known that the disease is preventable, and morbidity tables, in not a few localities, have already been per-

ceptibly influenced by the sanitary measures put in operation as a result of knowledge acquired within a few years. For a very considerable period yet to come, however, consumption will continue to rank as the most effectual weapon of the Grim Destroyer, and the ratio of about one death from this disease to seven who are removed from earthly joys and sorrows by all causes will not be materially altered until we can bring entire batteries to bear where as yet only occasional random shots are heard. If it became known that we were about to engage in a war with a great foreign enemy which would probably result in the sacrifice of the lives of 100,000, 50,000, or even 25,000, of our fellow-citizens, how quickly would our country spring to the call to arms. The great patriotic breast of America would thrill with heroic determination and resolve, and not a hamlet in our broad land that would not yield up its best and bravest in the national defense. Yet we are engaged in a perpetual war with an enemy far more deadly and implacable than any foe which may come against us with ships and soldiers and guns. At the current death-rate from pulmonary consumption, more than 10,500,000 of the present population of the United States must succumb to that disease. The paramount importance of this subject is, therefore, apparent to all. We know the enemy. We are acquainted with his origin, with his methods, and with his manifestations, and we are also fully cognizant of a well-tested and successful, yet withal, expensive means of operating against him. The important question is, do we possess sufficient good sense, and sufficient love for our fellow creatures to employ our knowledge to the best advantage? Prevention is our chief reliance for the future, but let us here consider the great problem; How can we best combat the progress of consumption after it has actually manifested its symptoms and signs in the patient's economy?

It is the writer's absolute conviction that some form of climatic treatment, with its accessory circumstances, offers far more hope than all other methods of treatment combined. In an experience of more than twenty years the writer has not known of a single recovery from undoubted tuberculosis in which a change of surroundings did not constitute an important factor. The patient should, as speedily as possible, be removed from the locality in which the disease was inaugurated. The appearance of phthisis is in itself an evidence that there is something wrong about that particular atmosphere for the patient in question, and if he remains under the same environment he is almost sure to get worse. In the incipient stage the writer has observed some improvement in a removal merely into another

street in the city limits of New York. Unquestionable good results have followed a transference of the patient's abode from points in Brooklyn and lower Manhattan to Washington Heights in the upper part of the city. The hills above Scarsdale on the Harlem road, the elevated ridges about Summit, Short Hills and Morristown, and other points in easy reach of New York have proved themselves of very good therapeutic value, not only in restoring lost tone and vigor in the feeble and anæmic, but in not a few instances, in arresting a plainly tuberculous tendency.

At very convenient distances from the city environs will be found localities possessing atmospheric qualities quite at variance with those existing on Manhattan Island. Prior to April, 1901, when Dr. Legrand N. Denslow presented his paper on the climate of Long Island to the New York Academy of Medicine, few medical men realized that within two or three hours' railway journey in an easterly direction from New York could be found an area possessing a climate from May to October almost identical with those to which, during the winter months, we send our patients on journeys of one thousand to three thousand miles. Let us for a few moments pause to consider some of these atmospheric conditions as shown by Dr. Denslow's observations. The highest temperature observed at Long Beach in July, 1900, was 88° F., the lowest 63° F., a range of only twenty-five degrees, or less than at any point in the United States for the corresponding period, except San Diego, California. The average relative humidity for August of the same year was sixty per cent., for September fifty-four per cent. This was a much lower average than was observed at any point along the Atlantic coast, lower even than at San Diego or Los Angeles. The average relative humidity for New York City is seventy-eight per cent. The three coolest points along the coast were found to be Nantucket, Block Island and Long Beach. Over a greater part of the State of New York the precipitation is heaviest during the summer months. The reverse would appear to be true for Long Island, at least along the south shore and at the easterly end. W. A. Fleet, the United States Government Observer at Cutchogue, states that at that point there are one hundred more clear days annually than at New York City, and one hundred and sixty-two more than at Rochester, New York. During June, July, August and September, 1899, there were but eleven cloudy days, while for the corresponding months of 1900 there were only seven. Fogs are comparatively rare. No satisfactory theory has been advanced to explain the remarkable meteorological differences between this area and points on the mainland less than a degree of longitude distant, but these great advantages of dryness,

equability and clearness, coupled with its proximity to large centres of population, would seem to mark this district as a promising refuge for certain varieties of bronchial and pulmonary troubles. Cases which progress favorably along our southern seaboard in the winter should certainly do well on Long Island during the hot months. During the past summer the writer ventured to recommend the easterly and southerly part of Long Island tentatively to three persons having trouble with the respiratory organs. One was a case of chronic bronchitis with asthmatic paroxysms; the second was a case of incipient pulmonary tuberculosis, while the third patient was well-advanced in the same disease. The results were as follows: The patient with bronchitis was markedly benefited, and there were only two or three slight asthmatic attacks during a sojourn of two months on the Island. The case of incipient tuberculosis was apparently arrested, there being a disappearance of the cough and physical signs, while the case of advanced phthisis appeared to jog along down hill at about the same rate it had shown in the city, the patient dying in December last.

Within a very short distance of New York City in almost the opposite direction will be found another favored locality possessing a moderately elevated, cool and dry inland climate, quite distinct from that above mentioned. The author refers to the Highlands of Southeastern New York, a small detached spur of the Alleghanies, of which the town of Liberty (2,300 feet elevation), near the dividing line of the Delaware and Hudson water-shed, may be taken as a representative point. By reason of the establishment of the large and flourishing Loomis Sanatorium, this region has become well known as a resort for tuberculous patients. The writer will not dwell upon its advantages further than to say that this district offers a combination of desirable features difficult to excel. Patients do well in Sullivan county all the year round.

It is quite remarkable what a transformation has taken place in our views regarding the effects of cold on tuberculosis. Only a few years back we were in the habit of sending all our patients who could get away, to the South with the first appearance of cold weather, quite regardless of the type of the disease. Experience has slowly taught us that many patients fare better at our northern resorts during the cold winter months than in the summer. As many as four of the writer's patients are at this moment sojourning in Sullivan county, where the snow is thick on the ground and the temperature many degrees lower than in the city, and the diurnal variations more marked. The distance being comparatively short, the patients are able to make occa-

sional visits to the city and can be kept under fairly close observation. All of these persons are doing well, and it is hoped that a permanent cure will result in each case. We have learned that a low temperature, especially if free from excessive humidity, is often much more inimical to the development and growth of the bacillus than it is to his intended victim. Nor does variability of heat or cold count for much, provided the atmosphere possesses the qualities of purity, dryness and brightness. There are many other favorable, though less known, localities in the Shawangunk, as well as in the Catskill Mountains, which will no doubt occupy prominent positions among the health resorts of the near future.

What has been said regarding Sullivan county applies with equal or greater force to the Adirondack region in Northern New York, barring the single element of greater distance from the immense population in and around the metropolis. This fine region, embracing as it does, about 10,000 square miles of territory, could afford sites for sanatoria, tents and camping grounds sufficient to accommodate all the tuberculous patients in our State, and still leave unnumbered acres for private reservations, gentlemen's hunting preserves, etc. The winters are long and cold in the Adirondacks, from seven to ten feet of snow falling annually, but the right kind of cases may safely remain all the year. According to Dr. Trudeau, than whom we have no better authority, the best results are observed during the cold months. It is not desirable in this place to attempt to detail the many advantages presented by this lovely and picturesque region. Suffice it to say that it offers a safe and hopeful retreat for many types of tuberculosis, and it will no doubt continue to grow in professional favor.

Meteorological records at many points in the United States are still very meagre, but even with our present incomplete and unsatisfactory knowledge, it is apparent that within our limits are contained the counterparts of all the most favored climatic areas of Europe. In moist, warm climates, in warm, dry and desert climates, in moderate elevations, and in high and dry mountain resorts, it is easy to show that the conditions within our own borders are in no wise inferior to the best found in the old world. Even in the single item of island climates, in which we may have been somewhat deficient, there is a strong probability that we have lately become fully supplied.

As practical experience accrues and climatic knowledge of our own country broadens, many innovations in professional practice must take place, and there will be a general rearrangement of our present ideas and methods. Many fine salubrious regions

have never yet been utilized, while a few have been decidedly overworked. Almost every state in the Union which possesses a mountain district with plateaus of 1,500 feet elevation or over will be found to have localities suitable as resorts for tuberculous persons. Such districts exist in the White Mountains of New Hampshire, the Green Mountains of Vermont, the Blue Ridge in Pennsylvania and Virginia, and the main ranges of the Appalachian chain extending south through the Carolinas and Tennessee. All of these states contain excellent examples of moderately high mountain climates. In Florida and along the Pacific coast of Southern California are found the very best types of marine climates. El Paso, Texas, represents a region possessing a superb elevated, warm, dry climate, while in Arizona we find the prototype of the much-vaunted Egyptian desert region. The great plateaus of the Rocky Mountain chain present typical examples of the best Alpine mountain climates, which attract health-seekers from all parts of the globe.

Just a word about two or three of these localities. The Florida peninsula has probably been more written about and more indiscriminately recommended as a resort for consumptives than any other state of the Union. Many tuberculous persons have been sent there or gone on their own responsibility, who would have been very much better off at home. This unwise influx of hopeless cases has perceptibly influenced the mortality-statistics, and materially damaged the reputation of the peninsula as a health resort. The writer is well aware, as a result of a recent tour of the state, that many persons still go there to the prejudice of their cases, while others render nugatory the benefits of the climatic conditions by entering too freely into the social gayeties at some of the new, fashionable resorts. It can not be gainsaid, however, that the climate of Florida is just as salubrious and as beneficial as it ever was in the treatment of phthisis, *i. e.*, phthisis of the right kind. It is undeniable that several of the quiet and peaceful localities of the peninsula, including Rock Ledge, Ormond and Daytona on the east coast, Winter Park and Kissimmee in the interior, and Tampa and Belleair on the Gulf, offer as favorable conditions for the residence of properly-selected phthisical cases during the winter months as are to be found on the globe.

The state of Colorado offers exceptional attractions as a resort for weak-lunged persons, or for those who are in the early stages of tuberculosis. Patients may safely remain in the state all the year, and there are opportunities for remunerative employment in the flourishing plateau towns which do not exist in any similar locality in the world. Many of the opulent citizens of

Denver at the present day went to the state years ago victims of tuberculosis. They live as speaking testimonials of what the Colorado climate is capable of. Yet the author knows of many bitter disappointments. A physician, himself at one time afflicted with early tuberculosis, stated a few weeks since that another month in Colorado would have killed him. He had just sufficient strength left to make the journey to Los Angeles, where he speedily recovered.

It should be borne in mind that there is something very positive about the bracing, stimulating, mountain air found at the high elevations of the Colorado resorts. Weak, delicate and nervous persons should feel their way very carefully, in going there, and in many cases it is better to pause at some intermediate point of less altitude before risking the higher elevations. (This subject will be referred to below.)

This brings us to the consideration of a most vital question in connection with the climatic treatment of tuberculosis. Does experience or reason supply us with any indications whereby we may with a fair chance of success direct a particular patient to the locality where he will be most benefited, *i. e.*, where the atmospheric conditions are most suitable for his case? Much remains to be learned in this direction. The empirical method is still largely relied upon, but we have come in possession of certain rational guidemarks which should always be taken into consideration in deciding the momentous question as to whether the patient should leave home, and when and where he shall go.

In the very beginning of the treatment it is highly important to act promptly. Recurring again to the subject of removal, it may be stated that as soon as the disease is identified the patient's environment should be changed unless there are special reasons which forbid this step. The fact that the bacillus has gained a foot-hold shows a co-relation between the environments of that particular locality and the patient's system favorable to continued bacillary growth and development. The disease is almost sure to progress. But in sending a patient away from home, it is not wise to rely blindly upon the climate alone. This should not by any means be regarded as a specific for phthisis, but only as one of our most highly valued aids. Perhaps the patient will not require so much in the way of drug treatment as at his home in the city, yet the much prized and highly useful pharmaceutical preparations employed in this disease should not be recklessly cast aside. Besides many of the symptomatic indications, no matter where the patient may go, will continue to require occasional drug treatment. Cases with much fever or other constitutional disturbance should always be treated temporarily at

home before sending away. With reference to where the patient should go, a consideration of the following points is useful:

(I.) Ordinary cases in the early stage with slight lung involvement and little constitutional disturbance usually show improvement with almost any change of climate. Such cases frequently recover at Liberty, in the Adirondacks, in Florida, Southern California or Colorado.

(II.) In catarrhal cases attended by profuse secretion, a dry climate should be chosen, preferably a warm, dry climate, with as little wind as possible. Atmospheric conditions in Arizona and in the neighborhood of El Paso, Texas, barring the occasional northers at the latter place, are very favorable for this class. Nearer home, Thomasville and Summerville in Georgia, and Aiken, South Carolina, are beneficial. Many catarrhal cases do well in Colorado and New Mexico. These patients usually fare better in moist climates.

(III.) Persons of the nervous, irritable or erethistic temperament do better in a marine climate and should not be sent to high elevations. Florida or Southern California may be chosen for the winter months; Old Point Comfort or Atlantic City for the spring, and, perhaps, the south shore and easterly end of Long Island for the summer.

(IV.) Sir Hermann Weber advises against sending cases of ulcerative tuberculous laryngitis to high elevations. It would appear that in Colorado not over twenty-five per cent. of such cases receive benefit. We are informed, however, that seventy per cent. of cases of this type show improvement at the Loomis Sanatorium at an elevation of 2,300 feet. The writer has seen excellent results in both Florida and Southern California.

(V.) Most chronic, slowly progressing cases, especially fibrous phthisis, do best in a marine climate. Such persons living in the East do well to spend the winter months in Florida, coming north by easy stages as the spring advances. Even in New York City and vicinity patients of this class often enjoy long periods of immunity from any particular symptoms. One lady under the writer's care, suffering from fibrous phthisis, passed the last twenty-five years of her life in comparative comfort between Orange, New Jersey, and Manhattan Island.

(VI.) Cases of phthisis complicated by heart disease should be kept at comparatively low levels. Persons with enfeebled circulation who suffer from cold hands and feet, and who shiver in slight draughts, should also keep away from high altitudes.

(VII.) Cases involving much loss of lung substance, or the withdrawal of any considerable portion of the respiratory area from the exercise of its functions, whether from compression of

the lung due to empyæma or simple pleurisy, to excavation, to retraction from old pleurisies or to empysema, usually fare badly at high elevations. Such cases are not susceptible of permanent cure, but their condition is considerably ameliorated by winter residence in Florida or along the Southern California littoral. Theoretically, the low-lying desert belt of Arizona should furnish an ideal climate for such patients, but experience with this region is so far too meagre to supply us with reliable data.

(VIII.) Puny youths, with transparent skins, spindle legs, and ill-formed chests, not actual, but potential, or probable cases of tuberculosis, are generally benefited by a prolonged residence in a high mountain region.

(IX.) There has been considerable difference of opinion in reference to a proper climate for hemorrhagic cases. The first patient ever referred by the writer to Colorado was seized with his initial pulmonary hemorrhage on the day of his arrival in Denver, and suffered daily from this cause during a three weeks' stay, when he returned East. But not a few subsequent hemorrhagic cases have thriven remarkably well at some of the high Colorado and New Mexican resorts. The most careful observers are apparently beginning to agree that the occurrence of hemorrhages is no bar to high altitudes. Dr. S. G. Bonney, of Denver, in a recent communication embracing a very large experience in Colorado, presents indubitable evidence to show that that elevated region is not prejudicial to hemorrhagic cases. It would appear with our present knowledge, that the tendency to pulmonary hemorrhages does not in itself furnish an indication either for or against any particular variety of climate.

(X.) It is urged by some writers that cases of phthisis accompanied by albuminuria or marked diabetes should be kept away from high climates. The writer, however, has been unable to find any satisfactory evidence in support of these views. Cases so complicated are usually of great severity and apt to be progressive. It would, therefore, appear to be unwise, as a rule, to send persons having these troubles far from home in any direction.

Let us now consider a final and very practical aspect of this question. We have a variety of the best climates in easy reach. The patient with the means and time at his disposal may have the advantage of any kind of atmospheric environment his medical advisor chooses for him. But how about that vast majority who are engaged in the struggle for their daily bread, and too often have helpless families depending upon them, and who must remain at their work as long as they can drag one foot after the other? It is not intended here to decry the magnificent

benefactions of our wealthy philanthropists to universities, colleges, museums of art, etc. These are grand and noble works and worthy of all praise and emulation, but they will be of little use to the thousands all over our fair land, over whom is hovering the grim spectre of tuberculosis, or who have already entered upon the death struggle with the fell destroyer. If such a person could at once suspend his occupation and be sent without delay to a properly selected locality in the country, whether on the seashore or inland, where he could for the time be free from the worry and care of toiling for his living, where good food and lodging may be had, where skillful medical attendance and nursing are supplied, and where the patient ceases to be a menace to his associates and the community, a long step toward the extermination of this scourge would be accomplished. It is the author's conviction that the rich man who establishes and endows one such sanatorium, where his suffering and dying fellow-creatures may be won back to health and hope and usefulness, is worthy of greater love and honor from his countrymen and will occupy a higher place in the Final Accounting than he who embellishes a costly church, builds a library, or founds a university.

Some of our French contemporaries have recently been passing the time by estimating the cost of providing suitable sanatoria for the half million consumptives in France. They have succeeded in staggering themselves out of all hope in the possibility of sanatorium treatment. It is, therefore, recommended that we concentrate all our energies upon prevention alone, which is practically an abandonment of the poor devil who already has the disease to his fate. I thank God, gentlemen, that advice proceeds from France and not from America. It has not been a habit of our countrymen to quail at undertakings because they were difficult and expensive. If we can succeed in fully convincing our law-makers, as well as our millionaires, of the *necessity* for tuberculosis hospitals, the means for their construction will be forthcoming.

Discussion

Dr. Alfred Meyer, of New York: We can all agree with the author, that there are great advantages to be obtained from the great variety of climates in our country. I am happy that he has said a good word for some of the near-by climates, but no mention has been made of one very convenient place. I wish to recommend to those living in New York city the use of the fire escapes on the tenement houses. I have recently treated some of the cases of tuberculosis in this way, and I have been surprised,

and I am sure others will also be surprised, at the results. There is just one objection to this treatment, *i. e.*, there is an ordinance against such use of the fire escapes. I have asked the fire department about this, and the reply was that "it was up to the building department." The building department said "it was up to the police department." I expect very soon to publish some of the results obtained in this way, and I am confident medical opinion will support us in violating that ordinance about obstructing fire escapes.

Dr. CROOK: I fully concur with Dr. Meyer concerning the value of this utilization of the fire escapes by our tenement house population, for, it is about the only form of climatic treatment that many of these people can afford.

XIV. MEDICAL SCHOOL INSPECTION IN THE CITY OF NEW YORK

BY HENRIETTA P. JOHNSTON, M. D.
NEW YORK

Mr President and Members of the Medical Society of the State of New York:

FOR many years the older physicians of New York City, who have made a special study of the diseases of infancy and childhood, had been investigating sanitary conditions in the public schools and arriving at definite conclusions with regard to the spread of contagious diseases among and by the pupils.

The responsibility of keeping a school free from this class of diseases rested upon the principals and teachers, many of whom, how much soever they might be interested in their own safety and the protection of the children under their care, were not capable of detecting symptoms which would be at once recognized by the practiced eye of a physician. In May, 1896, this subject was brought up for discussion before the Academy of Medicine and a committee was appointed by the president for further investigation of existing conditions and for devising ways and means by which daily examinations of all suspicious cases might be made by competent persons in whom could be vested the power of excluding from the school any child whose presence in the class-room might become a source of infection.

Dr. George B. Fowler, the chairman of this committee, in his report submitted a working scheme, which with modifications was subsequently adopted. In October, 1896, a communication was addressed to Mr. Charles Wilson, president of the Board of Health, by Dr. Charles Roberts, the Sanitary Superintendent,

"setting forth his belief that the transmission of contagious diseases among the children of this city was through their contact one with another in schools, and recommending daily examinations. As a result of all the combined forces, the Division of Medical School Inspection was organized by the Board of Health. The city was divided into thirty-five districts, and from the Civil Service list 150 men and women were selected whose duties were well defined by the Chief Inspector of the Division.

A widely distributed service and the assignment of schools whenever practicable to the inspectors residing in the school districts enabled them to make early morning visits, and all doubtful cases sent out by the teachers for examination were immediately excluded from the school if their condition justified such a procedure.

It was soon proved that the public schools had been regarded by many mothers as Day Nurseries, where indisposed children could be made more comfortable and receive better care than in their own homes; chicken-pox, measles, whooping-cough, and mumps, being looked upon as incident to child life not requiring special care or treatment and prophylaxis or isolation being unknown, excepting in those cases attended by a physician or reported to the Board of Health. In this initiative respect medical school inspection had been educative for parents as well as pupils, and the number of sick children deliberately sent to school may now be considered negligible, but it is safe to say that hundreds, if not thousands, of little children would be daily turned out of their homes and the doors locked at any early hour, and they would be compelled to remain all day in the streets while their parents are at work or engaged in trade but for the warmth and shelter offered by the public school, to say nothing of the protective care and the moral and intellectual training so freely given.

The total number of examinations made by the Medical School Inspectors during the first year of the service was 108,628. The total number of exclusions for all diseases found in the schools was 6,829. In addition to the school work, fifty of the inspectors were retained on "The Summer Corps," and, as in previous seasons, they were required during July and August to visit every family in every tenement house in the district assigned, to offer treatment to any sick person not receiving medical care, to advise mothers with regard to the care and feeding of their infants, to distribute tickets for the excursions of St. John's Guild, to give tickets for sterilized milk when needed, to reform whenever possible minor nuisances or report them to the Department of Tenement House Inspection. The following tabulated

statement will give an idea of the total amount of work accomplished by the "Summer Corps" during one heated term:

Number of visits to tenement houses	30,601
Number of families actually visited	274,742
Minor nuisances abated	4,057
Complaints of nuisances forwarded	539
Circulars for care of infants distributed	81,517
Revisits made to the sick	4,429
Patients under treatment by other physicians	5,402
Days' service rendered	2,159

In September, 1900, an epidemic of conjunctivitis made its appearance in the public schools. Over 100 cases were presented for examination on the opening day of one of the schools of the Children's Aid Society, and many more, who reported for admission only, were enrolled and then taken home to be placed under treatment. The disease, when promptly attended to, yielded readily to the boric acid wash and cleanliness, but in neglected cases the trouble continued for months, and in some instances became chronic. The total number of exclusions for eye diseases during this year was 6,258.

In the latter part of June, 1901, before the closing of the schools for the summer, sixteen oculists were appointed by the Board of Health for special eye examinations.

The report was as follows:

Thirty-six schools were visited and 55,470 pupils were examined.

Of these 6,670 were found to be afflicted with contagious ophthalmia.

2,328 of these were of the severest type of trachoma, necessitating almost immediate operation.

3,243 were of trachoma of the milder type.

1,099 were of acute conjunctivitis (purulent.)

It was the opinion of the examiners that all of these should be excluded from the school to prevent further infection.

The number of cases in the boys was noticeably greater than in the girls.

The percentage in boys 28% to3.6%.

The percentage in girls 18.6% to 1%.

In September, 1902, a new method of School Inspection was adopted by Dr. Ernst G. Lederle, the president of the Board of Health. This required daily morning examinations as before, and in addition to this a routine weekly examination of every child in every class, special care and vigilance to be exercised in examining the kindergarten and primary classes. This examination to include head, eyes, throat and hands. The increasing prevalence of eye diseases, especially among the pupils of

the lower East Side, became a source of alarm. The number of cases of trachoma excluded and placed under treatment, often operative, filled the dispensaries and eye hospitals with patients. At the Department of Health an oculist was employed for special examinations, and a portion of Gouverneur Hospital was set apart by the trustees of Bellevue Hospital for the use of the department for the children living and attending school in this neighborhood. From December 17, 1902, to January 11, 1903, inclusive (twenty-five days), 950 new cases applied for treatment, and 127 operations were performed. To test the intelligence of one class of Jewish boys, the writer asked if any one could tell what "trachoma" meant, and the word was written on the blackboard. One boy gave a very good description of the disease, and a bright-looking lad took out of his vest pocket a clipping from a daily newspaper, entitled "Trachoma, how to prevent and how to cure it." A third answer, not so scientifically correct, but extremely suggestive, was "A new kind of comb to make the hair clean."

The services of nurses in the schools have been very valuable, for the treatment of minor surgical cases, catarrhal conjunctivitis, ring-worm on the face or hands, etc., thus limiting the number of exclusions and at the same time securing attention for conditions which might otherwise be neglected. Pediculosis capitis, which has been the rule and not the exception in a large majority of the public schools, is treated by the nurses at the homes of the pupils, and the mothers are carefully instructed in the methods most successful in combating this disease. If these directions are not obeyed a second visit is made, and if after this the children return to the school unimproved they are excluded until their condition warrants a formal re-admission by the inspector.

A very interesting question arises in this connection with regard to the relation of health and life to hygienic surroundings.

On the top floor of a rear house in Pitt street, in one rather small living room, the writer saw in an apparently well-preserved condition the female representatives of four generations, in an ascending scale, the child, the mother, the grandmother and the great-grandmother. The school children on the lower East Side are for the most part rugged and healthy in spite of the fact that their homes are, many of them, overcrowded, poorly ventilated and unclean. They are often scantily clad and can be seen any stormy day, even in mid-winter, running to and from the school without any hats, umbrellas or overshoes. One little girl, who was sent home by her teacher because her dress was not clean enough to be allowed to remain in the class-room,

returned in about an hour with it wet on her shivering little body, her mother having in the meantime washed it. The child was sent home again with very explicit instructions as to the treatment she should receive from her mother and they could not well be disregarded. Thus through the medium of the school children are many wholesome lessons conveyed to careless, ignorant or indifferent mothers. As a further illustration of their habits is the fact that many of the children have their clothes sewed firmly on in the fall, not to be removed under any circumstances until spring, and it is not an infrequent occurrence to find four or five dresses on a child, added one over another as the cold weather comes on, and these cumulative garments are kept on night and day. One of the principals who had been for a number of years in charge of a large school in this congested quarter of the city says: "It is a survival of the fittest. A child who lives to be old enough to be admitted to the public schools is as tough as a pine knot." It is quite certain that the weaklings die in great numbers. The infant mortality for the year 1901 was in the Borough of Manhattan, 13,421. In October, 1902, in examining thousands of throats, many hypertrophied tonsils were noted, not one in a state of acute inflammation, and only two cases of post nasal catarrh. From September to December, 1902, inclusive, the total number of examinations made by the school inspectors was 2,991,872. The total number of exclusions was 41,240. For the study of child character, both native and foreign, and for the study of racial peculiarities, the work of the inspector is most favorable. In Greater New York, as elsewhere, the tendency to colonization is strikingly marked. In one school district the attendance may be exclusively Jewish, Polish, Russian, and it is surprising to see how rapidly the children acquire a new language, for many of them cannot speak one word of English when they enter the school. The "Jargon" or "Yiddish" is the language of the home and the neighborhood, and the adult members of the family are often compelled to depend upon the children, who act as interpreters for them, in all the ordinary transactions of a busy life. The children of Jewish parents show in a pathetic way the centuries of persecution to which the race has been subjected. They are not resentful of discipline, and they are submissive to reproof and punishment. It is difficult, if not impossible, to make them answer any questions which will incriminate them, and the old Spartan law is carefully observed. They are indefatigable in their efforts to acquire an education, and the ambitious parents never seem to allow an opportunity to be lost, often limiting themselves to the bare necessities of life that they may educate a

son in one of the professions or a daughter as a teacher. In a house in Lewis street, on the ground floor in the rear of a meat market, a young man was studying in a room full of people. He was a student in the Bellevue Medical College, preparing for his examinations, and is now a practicing physician. In another room, under equally unfavorable conditions, were two sons, one a student at the College of Physicians and Surgeons, and the other at the College of Pharmacy, and many of the prizes even at the universities are awarded to the Jewish competitors. Added to all this, their peculiar beliefs and religious rites are never allowed to suffer neglect, and the boys are compelled after the public school hours to attend a Hebrew school to keep inviolate their own language and customs. If the father should die, the eldest son assumes all this paternal responsibility and conducts with great solemnity the religious services at the family altar.

The Italian children are of the musical and artistic temperament. Some of their art work is quite remarkable. Their singing voices are sweet and melodious, and they play naturally on musical instruments. They are excitable, and panics are likely to occur among them, spontaneously, as it were, unless the teachers are watchful. As a rule, their habits are exceedingly unsanitary. Concerning diseases of the scalp, they have a superstitious notion that they must not be treated medically, and favus is called "Holy Soil." Consequently these diseased conditions are sadly neglected until the children apply for admission to the school and are handed over to the inspector for examination and exclusion.

In another quarter of the city the school population may be mostly Irish. American children are not numerous, and colored children are conspicuously absent from the so-called white schools.

These colonies are not going to remain on the lower East Side. They are migratory, and are already moving into the more ambitious residential quarters in the various boroughs. A certain section of Upper New York is topographically described as "Little Italy." Nor can it be said of these people, "Three generations from shirt sleeves to shirt sleeves." Their cycles are of indeterminate length, and the Jews are emerging from a bondage to which they will never return. The immigrant people, who are most likely to influence the destiny of Greater New York and, therefore, the most interesting, are the Jews. The Alien steerage arrivals at the port of New York for the year 1902, as computed by the United States Immigration Authorities at Ellis Island, were 551,569 passengers as against 412,416 passengers in 1901.

The largest recorded number for a single year since the Bureau of Immigration was established in 1819.

The Federal laws, while admirable in many respects, should be made more drastic in *this*.

No immigrant should be allowed to land upon our shores whose sanitary condition would jeopardize the public health, and a physical examination should be made by the Federal Authorities, not superficially, but in the same thorough manner as that which has been adopted by the Board of Health of this city for the examination of the children in the public schools and in the homes. If these measures were persistently enforced as a matter of public policy we would be able gradually to eliminate the uncleanly and the diseased from among the immigrants, for those people intending to emigrate to America would prepare themselves for the trans-Atlantic voyage, knowing the difficulty of effecting a landing in this country unless the sanitary requirements were fully complied with. Time is needed for such local reform measures as have been and may be inaugurated by the Department of Health of New York City—the condemnation and demolition of whole blocks of old and filthy houses to create large spaces for parks, large play-grounds for little children, large resting places during the cool evening hours for weary men and women who have been toiling in the heat of the summer sun, large breathing spaces for all who come under their benign influence. Can this be accomplished without time, the great factor in all the Reform Movements of the world's history? Time is needed for the Board of Education to enforce its laws compelling all children to attend school, and protecting children from engaging in mercantile or other employments until they have had the benefit of a common school education. Time is needed for the work of its finely equipped night schools for men and women, offering education in the languages, the trades and all the English branches, without money and without price.

With these splendid opportunities for physical and mental development, all foreign people who are permitted to land upon our hospitable shores might in time become well adapted to the new conditions in which they find themselves in our glorious country and be made to contribute generously to its welfare.

XV. PUBLIC HEALTH AND THE MARINE HOSPITAL SERVICE OF THE UNITED STATES

By DANIEL LEWIS, M. D.
NEW YORK

Mr. President: When you honored me with the suggestion that I should discuss the advisability of the establishment of a national board of health, or the appointment of a secretary of public health, who should be a cabinet officer, you will recall the fact that I expressed a doubt as to the possibility of securing legislation favorable to either project.

It is not my purpose now to enter into a discussion of the advantages or disadvantages pertaining to either plan. There can be no question that the repeated demand for such a federal supervision of the public health and sanitation as was contemplated, sprang from a deep conviction of its necessity in the minds of many physicians in the United States. The American Public Health Association has several times passed resolutions favoring the establishment of a national board and, if I am not mistaken, the American Medical Association has declared in favor of a secretary of public health as a cabinet officer.

The difficulty, from the commencement of this agitation—which has continued since the former national board of health ceased to exist, through lack of appropriation—has been, the impossibility to enlist the interest of Congress in either proposition.

The conviction has grown into a practical certainty that whatever could be secured in the way of a national health organization, must be a natural outgrowth of the Marine-Hospital Service. It, therefore, naturally followed, that when the Surgeon-General of the Marine Hospital Service had a bill prepared about one year ago, which decidedly increased his powers in regard to the conservation of the public health, and invited the representatives of state boards of health and quarantine officers to meet him in conference, that there was a practical unanimity in favor of the amendment proposed by him, which finally passed Congress, being approved by the President on July 1, 1902.

The provisions of the new law—familiar in a *general* way to the members of the Society—may not have received the *careful*

consideration of many of the members present, for which reason, I take the liberty of mentioning a few of them. It may be stated, by way of introduction, that the powers originally conferred upon the United States Marine Hospital Service are continued under the new law, which establishes the Public Health and Marine Hospital Service of the United States. The Surgeon-General in charge of the Marine Hospital Service becomes the Surgeon-General of the *Public Health and Marine Hospital Service,* with a salary increased from $3,500 to $5,000; and the same rules relating to the detail of army and navy officers obtains under the new law, as under the former one. The essential differences, however, are these:

Section 5 provides—that the Hygienic Laboratory, which was established by act of Congress, approved March 3, 1901, under the jurisdiction of the Marine Hospital Service, shall hereafter be under the direction of an Advisory Board of experts in pathological and bacteriological and general laboratory work, who shall be appointed by the Surgeon-General, being approved by the Secretary of the Treasury. The five members comprising this board shall each receive compensation of $10 per diem while serving in conference with the Surgeon-General (also the director of the laboratory), as well as necessary traveling and hotel expenses. The term of service of each member of the Advisory Board will ultimately be five years, the original appointees being divided into terms of one, two, three, four and five years. The Surgeon-General has already appointed the following Advisory Board of the Hygienic Laboratory: William H. Welch, Simon Flexner, Victor C. Vaughan, W. T. Sedgwick, F. F. Wesbrook.

The eminent fitness of each of these gentlemen for the position is a guarantee to the profession and the public that the highest possible standard of efficiency will be maintained in the Laboratory work in the future.

The next important change secured by the new law is that provided for in Section 7, which reads as follows:

"That when, in the opinion of the Surgeon-General of the Public Health and Marine Hospital Service of the United States, the interests of the public health would be promoted by a conference of said service with state or territorial boards of health, quarantine authorities, or state health officers, the District of Columbia included, he may invite as many of said health and quarantine authorities as he deems necessary or

proper to send delegates, not more than one from each State or Territory and District of Columbia, to said conference: *Provided,* That an annual conference of the health authorities of all the states and territories and the District of Columbia shall be called, each of said states, territories and the District of Columbia to be entitled to one delegate: *And provided further,* That it shall be the duty of the said Surgeon-General to call a conference upon the application of not less than five state or territorial boards of health, quarantine authorities, or state health officers, each of said states and territories joining in such request to be represented by one delegate."

A careful study of this section reveals the fact that as thorough a co-operation with the existing sanitary authorities in the different states has been provided for as could possibly be secured under any national board legislation which has ever been proposed; and, in my judgment, the great need of a national authority to co-operate with state and local authorities has been adequately secured by this provision.

The first conference under this section has recently been held in Washington, the subject under discussion being "The Plague in San Francisco." Nineteen different state boards of health and other sanitary organizations asked for a conference, and all attended the meeting. These special meetings may be called, as you notice, at any time, whenever, in any part of the country there may arise a condition which seems to call for co-operation with the federal authorities; and once each year, each state sends a delegate in conference with the Surgeon-General of the Public Health and Marine Hospital Service. Under the law, the President is given full power to prescribe rules for the conduct of the Public Health and Marine Hospital Service to meet special requirements at any given time or place. And every state board of health and every state quarantine establishment, and terriorial government, including the District of Columbia, can at any time have a voice in the direction of sanitary rules and regulations and their execution.

I am firmly convinced that we have secured in this enactment a great advance over previous conditions, and that whatever has been gained will be a permanent gain, and the way remain clear for any amendments or modifications which, when recommended by the sanitary authorities, may be readily secured from Congress.

I bespeak for this new act, the hearty co-operation of the mem-

bers of this Society, and the suspension of the agitation for a national board of health, or the appointment of a medical cabinet officer, until the outcome of the new order of things shall have been amply determined by a few years' experience.

XVI. STERILIZED MILK, PASTEURIZED MILK OR CLEAN MILK

By DR. C. W. M. BROWN
ELMIRA

This paper is not read with the expectation that it presents much that is new, either in theory or in fact; but rather with the hope that by reiteration of well-known theories and facts, it may help somewhat to stimulate the profession to greater activity in the endeavor to secure clean milk.

As long ago as 1878, some attempt was made to improve the quality of milk in connection with German milk cure establishments.[1] *The Archives of Pediatrics* began publication in 1884, but not until 1888 does its index contain reference to sterilization of milk. In 1889 Jacobi,[2] who had long practiced and taught the wisdom of boiling milk for infant feeding, makes reference to the introduction of Soxhlet's apparatus by Dr. Caille as an improvement. But to Soxhlet belongs the great honor of having systematized and popularized the boiling or sterilization of milk for the special use of infants. Sterilization was soon accepted all over the world as a desideratum long sought in infant feeding;[3] "came to be looked up to as an object of faith, as a pope, among foods, infallible." Gradually through the years its approximate value has become more fully known, while its dangers and limitations have become more evident. The term "sterilization" is widely and rather loosely used to signify the heating of milk for the destruction of germs. It should be borne in mind, however, that none of the methods commonly employed renders milk sterile, in the bacteriological sense of the word. This may be done by heating on two or three successive days to 212 F., the so-called Tyndallization. What is accomplished is the destruction of the germs of typhoid and scarlet fever, diphtheria and tuberculosis with a large number of other bacteria, so as to retard for a time the ordinary fermentative changes.

It is conceded by all that one of the more important conditions of the infant's diet is that it should be sterile. Should the food be more difficult of digestion, or involve a longer time in digestion than its natural aliment, so much the greater is the necessity. Cows' milk, the most important substitute for human milk, has been obtained sterile under special conditions, but commercially, never is so, having from a few hundred or thousands of bacteria per cubic centimeters when obtained and kept under proper conditions to several millions when unhygienic conditions obtain or it has not been quickly cooled and kept so.

It should always be distinctly understood, that the application of heat to 212 F. will not make foul milk good and safe for food. Such milk still contains the dead bodies of the bacteria and the toxins derived therefrom. Conn has isolated and grown more than two hundred varieties of bacteria which he found in milk. Most of these varieties are seldom met with, others are common, while a few are nearly always found. Ordinarily the great majority are lactic acid producing bacteria, which chiefly come from the fore milk, being found in the milk ducts and cisterns of the cow's udder. Others still, according to Moore and Russell, produce alkaline or other forms of fermentation, and are largely from the dust of the stable, loose hairs and excreta from the cow's udder and flank. These are apt to be spore bearing and thus resist sterilization. While some bacteria are of great value to the butter and cheese maker for the development of flavors, so far as known none are of advantage in milk used for food, while others are decidedly poisonous. Milk raised to the temperature of 212 F. is markedly altered both in taste and smell, and many observers notice the changes which occur both in its chemical and physical properties, as well as in its digestibility. I quote these changes as summarized by Judson and Gittings[4] from many sources:

(*a.*) Decomposition of lecithin and nuclein.

(*b.*) Organic phosphorus is diminished and unorganic phosphorus is increased in amount.

(*c.*) The greater part of the phosphates are rendered insoluble.

(*d.*) Precipitation of the calcium and magnesium salts.

(*e.*) The greater part of carbon dioxide is driven off.

(*f.*) Normal lactic acid fermentation is prevented, allowing more deleterious fermentations to follow.

(*g.*) Lactose is destroyed. Johannesen says, however, that this does not occur below 230 F.

(*h.*) Caramelization of a certain portion of the lactose.

(*i.*) The fat emulsion is rendered imperfect or destroyed by coalescence of the fat globules.

(*j.*) Separation of the serum albumen begins at 167 F.

(*k.*) Casein is rendered less easy of coagulation by rennet.

(*l.*) Casein is slowly and imperfectly acted upon by pepsin and pancreatin. Leeds says, the proteid substances become attached to the fat globules and probably hinder to some extent, fat assimilation.

(*m.*) Peptone and toxins can be found after prolonged sterilization. [5]Vaughn says, that the toxin of the colon bacillus, which is constantly present and in abundant number in unclean milk, is not lessened in toxicity by a temperature of 356 F. One two hundred and seventieth of a grain will kill a guinea pig.

(*n.*) The starch liquefying ferment is destroyed and coagulated.

(*o.*) The taste is rendered objectionable and the cream does not rise well, although this does not lessen its value as an infant food. In explanation of some of these results Blackader[6] quotes: "Wroblewski, who points out that certain of the calcium salts which in normal milk are in a soluble state are made to enter in an insoluble combination by high temperature; the part played by calcium salts in connection with these ferments and their products whose presence is a special feature of organic fluids, is becoming more and more fully recognized." Duclaux had pointed out recently, that ferments of many kinds are only effective in the presence of minute quantities of calcium, magnesium or other mineral, the mineral varying with the special form of fermentation. For the coagulation of blood, calcium must be present, and for the coagulation of milk in the stomach, calcium in a more or less free form must also be present. If the calcium salts in the stomach through heat undergo alteration so that they are rendered insoluble, then the coagulation of the caseinogen will be to that extent arrested or delayed. In corroboration of this, it is found that outside of the body, boiled milk undergoes coagulation with rennet with much difficulty. Inasmuch as the primary coagulation in the stomach appears to be necessary for the normal digestion and absorption of milk into the system, it is certainly questionable whether boiled milk, as a rule can be absorbed and assimilated as readily as milk which has not been brought to a temperature sufficient to change the

condition of its calcium salts. It is probable that certain ferment-like bodies, which are of more especial value in the body, are found in milk according to experiments made by Babcock and Russell, of the Wisconsin Experiment Station,[7] who found that milk obtained in a sterile condition was capable of undergoing self-digestion because of the presence of a trypsin which was easily destroyed by heat. Holt[8] says, that "many of these changes are imperfectly understood and some are doubtless without any injurious effect upon nutrition." There is, however, one important clinical reason for believing that the nutritive properties of milk are impaired by heating to 212 F., viz: the occurrence of scurvy in infants, who are fed on such milk for a long time. Of 379 cases of scurvy brought together in the report of the American Pediatric Society in 1898, sterilized milk was the previous diet in 107.

To obviate many of the disadvantages enumerated, the practice of low temperature sterilization or pasteurization has come into frequent use in America, largely owing to the work of Dr. Freeman, of New York, and Russell, of the Wisconsin Agricultural Experiment Station. At first a temperature of 167 to 180 F. was used, and more recently a lower one, 140 to 160 F., for reasons which will be given below.

Considerable latitude with reference to temperature limits is permissible in pasteurization, but there are certain conditions which should be met, and these in a sense fix the limits employed. Inasmuch as it is not desirable to have any material change in taste and appearance in milk from that normally found in the raw product, the pasteurizing temperature should be limited to a degree of heat that can safely be employed without any danger of imparting a cooked or scalded flavor to milk. If the exposure is brought for any considerable time, say fifteen or twenty minutes, the change in taste appears to be quite prominent when the milk is heated to 158. This condition, therefore, determines the maximum limit which should be used in pasteurizing, if one is to avoid the production of the cooked flavor. Even below this temperature a slight change in flavor occurs, although it is destroyed upon chilling the milk. To be of value in increasing the keeping quality of milk and to insure freedom from disease bacteria, it is necessary in all cases to exceed the thermal death point of at least the actively developing bacteria in the milk. For most bacteria this limit is constant and quite sharply defined, running

from 130 to 140, where the exposure is made for ten minutes. Where exposed for a briefer period of time, the temperature limit is necessarily higher. The organism that is invested with most interest in this connection is the tubercle bacillus. On account of its more or less frequent occurrence in milk and its reputed high powers of resistance, it may be taken as a standard in pasteurization. Concerning the exact temperature at which this germ is destroyed, there is considerable difference of opinion. Part of this arises from the difficulty of determining exactly when the organism is killed, and part from the lack of uniform conditions of exposure. The standards that previously have been most generally accepted, are those of De Man, who found that thirty minutes' exposure at 149 F., fifteen minutes at 155 F., or ten minutes at 167 F., sufficed to destroy this germ. "More recently it has been proven and these results confirmed by different investigators that if tuberculous milk is heated in closed receptacles where the scalded surface pellicle does not form, the vitality of this disease germ is destroyed at 140 F. in fifteen or twenty minutes. If the conditions of heat are such that the surface of the milk is exposed, the resistance of bacteria is greatly increased. When heated in open vessels, Smith found that the tubercle was not killed in some cases where exposure was made for at least an hour." Considerable difference of opinion, as has been noted, has existed in the minds of medical men as to the relative digestibility of heated and raw milks, and considerable amount of experimental work has been done by making artificial digestion experiments with enzyms, also digestion experiments in animals and also in a few cases with children. The results obtained by different investigators are quite contradictory, although the preponderance of evidence seems to be in favor of the view that heating does impair the digestibility of milk; especially, if the temperature attains the sterilizing point. Those who wish to follow this subject further, are referred to the bulletin of the Maryland Experiment Station for August, 1901, by Doane & Price. These objections do not obtain in milk heated to a moderate temperature as in pasteurization, although this temperature, says Russell, lessens slightly its digestibility. The normal souring change in milk is due to the predominance of the lactic acid bacteria, but as this organism as a class does not possess spores, they are readily killed when heated above the thermal death point of the developing cell. The destruction of the lactic forms

leaves the spore-bearing types possessor of the field, and consequently, fermentation changes in heated milk are not those that usually occur; therefore, all heated milk, whether pasteurized of sterilized, should be cooled as quickly as possible after heating, and if used by children, should be consumed within twenty-four hours. If left under conditions favorable to germination, bacterial growth will go on, and as it has been shown by Flügge, the type of fermentation produced may sometimes be deleterious. The following experiments by Marshall[10] are of interest in connection with this point, as showing the influence of cooling on the germination of spores. Cultures of organisms that had been isolated from pasteurized milk were inoculated into bouillon. One set was left to grow at room temperature; another set was pasteurized and allowed to stand at the same temperature, while another heated set was kept in a refrigerator. The unheated cultures at room temperature showed evidences of growth in thirty trials in an average of twenty-six hours. Twenty-nine heated cultures at room temperature all developed in an average of fifty hours; while the heated cultures kept in the refrigerator showed no growth in forty-five days, with but four exceptions. It follows from this that after milk is pasteurized it should be kept in germ-free receptacles. From what has been said it would seem that the argument is altogether in favor of pasteurization. The lowest temperature and the shortest time that will surely destroy the objectionable bacteria in milk would seem to warrant general adoption. Pasteurization, however, requires more care, intelligence and more special apparatus, and if it is not properly done, it may be worse than nothing. Sterilization at 212 F. is simpler and may be done with many and inexpensive forms of apparatus, or without any apparatus. Therefore when no ice is available, it is certainly safer in hot weather than pasteurization. In cities and among the poor, milk heated to 212 F. for thirty to sixty minutes is probably not only the most satisfactory, but most efficient method of sterilization. In fact, this may be done two or more times a day. The risks sometimes attendant upon it should not be forgotten, especially when it is continued for a considerable length of time. It is important that sterilization be done at the earliest possible moment, before many bacteria have developed, because the fewer spores and spore bearing bacteria, which the milk contains, the more effective will be the sterilization, therefore the cleaner the milk the better will be the

sterilization. The clinical test, after all, is the court of last resort, in deciding what is best in foods or methods of feeding. The true value of pasteurization in large cities, is best shown by the great lessening of the mortality rate among infants in New York following the free distribution of pasteurized milk to the poor from the Nathan Strauss milk depots. That fresh, clean, unadulterated cows' milk, from healthy animals and free from pathogenic organisms is the best food for infants, and should be an important part of the food for growing children, all will agree; that it is one of the chief foods for those who are sick in later life, is also true. That such milk can be obtained is also true. And it is also strangely and unfortunately true, that it is only provided to the larger cities where the problem of distribution from dairy to consumer is so much greater than in smaller cities and towns where the time from production to consumption is much less. It is then to the proposition that such a condition of things is not necessary and should not be, that I invite your attention this evening. Well might Jacobi, the Nestor of American pediatrics exclaim last summer at Saratoga, that the greatest advance in pediatrics made in the last ten years had been the obtaining of pure, clean milk as the food for infants. So far as I know the Walker-Gordon Company supply eighteen cities principally in the United States, with good milk. The Milk Commission of the Medical Society of the County of Essex, New Jersey, control the Fairfield Dairy Company, which is the forerunner of all producers of good milk. Besides which the Milk Commission of the Medical Society of the County of New York issue certificates to several dairies for the City of New York, the Milk Commission of the Philadelphia Pediatric Society to several in Philadelphia, and more recently the Milk Commission of the Medical Society of the County of Kings has begun to issue certificates to others in Brooklyn.

In March of last year, agitation was begun in our Academy of Medicine for better milk. A milk commission of six physicians was appointed under its auspices. A bacterial standard of ten thousand per cubic centimetres and from three and one-half to four and one-half per cent. butter fat was established. Then a search for a dairyman was begun. No one was found ready and willing to comply with the requirements. Finally one dairyman was discovered who was willing to build new barns to endeavor to comply with the demands of the Commission, and in two or three months it is expected the Commission will be ready to issue

certificates. What has been done in one town of forty thousand, can be done in others, if the physicians desire it. What constitutes clean milk? There is no absolute standard. The number of bacteria present depends upon care as to cleanliness and control of the temperature and upon the age of milk. If milk is quickly cooled to 40 or 45 degrees, bacterial growth is slow. If great care is used as to cleanliness, which means a clean stable, free from dust, clean healthy cows, clean healthy milkers, clean utensils, the bacterial count will be low. The observations of Park,[11] and Hunziker[12] have shown that the germicidal action of milk lessens the number of bacteria during periods varying from six to twenty-four hours. Such milk will have almost as low a bacterial count in twenty-four hours as when drawn. This is not true, however, if care is not taken to exclude filth and to quickly cool it. The Milk Commission of the Philadelphia Pediatric Society have a standard of ten thousand bacteria to the cubic centimetre; New York and Brooklyn thirty thousand. Some special farms have reported as low an average as 2,250 through the year, others five, six and seven thousand, up to fifty thousand. The great value of these figures is in the strict cleanliness which must be used to get them. Much stress has been laid upon the necessity of throwing away the first few streams of the fore milk, because the fore milk contains a much larger number of bacteria. But according to the experiments of Hastings[13] in Russell's laboratory, this lessens its keeping quality. He found that the fore milk, though much richer in bacteria than the whole milk, does not coagulate nearly so soon. In the series of five trials the fore milk did not curdle at room temperature on an average until after eighty-four hours, while the average time of curdling of the whole milk was thirty-eight hours.

All cows should be tested for tuberculosis, and all those re-acting rigorously excluded, not alone because of the possibility of infection from the tubercle bacillus, but because, if the observations of Jemma[14] are confirmed, milk containing tuberculous germs is dangerous, even if strictly sterilized. He found that young rabbits fed upon sterilized milk containing the bodies of dead tubercle bacilli, died within fifteen or twenty days of marasmus. Other rabbits fed on plain sterilized milk free from tuberculous bacillus flourished. The autopsy showed only enteritis, and fatty degeneration of the liver.

It has usually been urged that common cows, ordinary varie-

ties, are less liable to tuberculosis than high-bred breeds. This may be true. But some dairymen have learned at tremendous cost, that a cow to do work must not only be well fed, but must be placed in good hygienic conditions otherwise. The air space must be abundant and the barns must be kept clean. Such cows so kept have little tendency to tuberculosis. A magnificent illustration of this is to be found at Briarcliff, where Mr. Law says he has no tuberculosis in his great dairy of high-bred Jerseys. If milk is unadulterated the butter fat will average about four per cent, and this may be taken as a possible standard in that respect.

It has been urged that milk produced under such conditions costs so much as to be beyond the reach of the average family. Both Chapin[15] and Pearson[16] say such milk can be produced at an average increased price of one cent per quart over ordinary milk.

Briefly then in summary. 1. Sterilization at 212 F. is of great value, especially in cities and to the poor who lack intelligence, because it may be performed by any one and with simple or no apparatus.

2. Pasteurization at a temperature of 140 to 158 F. in closed vessels for fifteen minutes is much to be preferred; as the milk is little changed in its taste or chemical properties from raw milk, and this temperature is sufficient to kill the pathogenic organisms and lactic acid producing bacteria. But we must agree with Holt[17] when he says, "that all heating of milk sufficient to kill bacteria, does impair to some extent its nutritive properties, and to a degree proportionate to the height of temperature employed and the length of time it is continued."

3. When obtainable, fresh, pure, clean milk used raw is much to be preferred.

4. It is now supplied to many of the large cities.

5. There is a strong and rising demand among the laity for such milk.

6. It can be provided in all cities and towns of even moderate size if the profession will put forth proper efforts to procure it.

7. Under a general law all milk dealers should be licensed by the city or town in which they sell milk. Such license should give the health officer power to enter upon and inspect the premises upon which the milk is produced at any time he sees fit, and should also carry with it a statement on the part of

the dealer that all cows producing milk which he sells, have been tested for and found free from tuberculosis.

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XVII. EXAMINATION OF MILK BY THE GENERAL PRACTITIONER

By HENRY LARNED KEITH SHAW, M. D.

ALBANY

Clinical methods for the examination of the patient and his excretions are now largely employed in the daily practice of physicians. The examination of urine, sputum, blood and stomach contents can be made in a short time, and medical students are well drilled in the technique of these methods before leaving the medical school and can deduct correct conclusions from their examinations.

In this paper it is purposed to outline a rapid and simple method of examining either human or cow's milk. There is a very prevalent idea abroad that a milk analysis requires expert chemical skill, expensive apparatus and much time. For these reasons the general practitioner rarely, if ever, attempts to determine the character of the milk from a mother whose baby does not thrive. As a result the child is weaned from the breast and some much-advertised food is prescribed.

It is not the object of this paper to discuss the importance and value of milk examinations, but it can be said, that it is impossible to feed infants scientifically or intelligently without making, or having made, milk examinations from time to time.

A practical examination for a physician must be easy of application, consume little time, require simple and inexpensive apparatus, and be accurate in order to reach reliable conclusions. The scheme I wish to present does not take much more time than a complete chemical uranalysis, and requires no more skill. No originality is claimed, for "I have gathered a bunch of other men's roses, and only the cord that binds them is my own."

Care must be taken in obtaining the sample of mother's milk to remove all the milk from the breast. The composition of the fore milk differs from the last portion withdrawn. The sample of cow's milk should be well mixed before testing.

The first step is to determine the total acidity of the milk. With cow's milk, this determination is of vast importance. Milk with an acidity over sixteen hundredths of one per cent. should never be used for infant feeding. The limit for drinking purposes is two-tenths of one per cent., and three-tenths of one per cent. is the point of souring. There is a close and definite relation between the degree of acidity and the number of bacteria. Souring is due to lactic acid producing bacteria, and milk containing many bacteria will have a high acidity. In this way we get some idea of the cleanliness employed at the dairy and of the care taken in the transportation and delivery of the milk. There is a very practical bearing to us as physicians, for "milk on the point of souring is very liable to produce gastric and intestinal catarrhs of an acute or chronic character in infants, children or persons with feeble digestion." The chief causes for an untimely acidity of the milk are improper feeding of the cows, dirty milk pans and rooms, unclean udders and teats, and a high temperature.

The articles required in making this test are few and consist of an alcoholic solution of phenophthaline, deci-normal sodic hydrate solution or a saturated solution of lime water, a small glass beaker, medicine dropper, and a small glass cylinder holding ten cubic centimeters and graduated into tenths of one cubic centimeter.

Five cubic centimeters of the milk are measured into the beaker and a couple of drops of the phenophthaline solution added. The cylinder is then cleansed and filled to the ten cubic centimeters mark with the alkaline solution. A small amount of the solution is taken up by the medicine dropper and added, drop by drop, to the milk which is well stirred with a glass rod. When a faint pink color is obtained the excess of alkaline solu-

tion is returned to the cylinder and the amount of the alkaline solution used is readily ascertained.

The following formula is employed when the sodic hydrate solution is employed:

$$\frac{\text{c. c. alkali} \times .009}{\text{c. c. sample taken}} \times 100 = \text{total acidity.}$$

For example, if it takes eight-tenths of one cubic centimeter to neutralize five cubic centimeters of milk, the equation would read:

$$\frac{.8 \times .009}{5} \times 100 = .145\%.$$

A saturated solution of lime water is approximately a one-twentieth normal solution, and when this is used the formula would read as follows:

$$\frac{\text{c. c. alkali} \times .0045}{\text{c. c. sample of milk}} \times 100 = \text{total acidity.}$$

A somewhat simpler but not so accurate a method is to use the Farrington alkaline tablets. These tablets consist of a definite quantity of alkali combined with coloring matter. These tablets keep much better than the standard solutions, require no apparatus, and are very cheap. The test is made as follows: One tablet is dissolved in a half ounce of water. Two drams of this solution are added to a like quantity of milk. If the resulting mixture, after shaking, is pink, the milk contains less than two-tenths of one per cent acidity. If the milk remains white, it contains more than two-tenths of one per cent. and is too sour to use even though it smells and tastes sweet. Other combinations can be tried, such as adding one dram of the solution to two drams of milk. A resulting pink color would indicate an acidity of less than one-tenth of one per cent.

A standard deci-normal alkaline solution can be made by dissolving twelve of these tablets in fifty cubic centimeters of water. When this is used for titration it is unnecessary to add phenœphthaline to the milk.

The *specific gravity* of the milk is next ascertained. For this purpose there are two types of lactometers. The New York Board of Health lactometer does not give the specific gravity of the milk directly, but has a scale divided into 120 equal parts, the mark 100 being placed at the point to which the lactometer sinks when lowered into milk of a specific gravity of 1.029 at 60° F. This lactometer requires a large amount of milk and certain calculations to convert the reading into the specific gravity.

The other type of lactometer is the Quevenne, which has the same scale as the ordinary urinometer and registers the specific gravity directly. These lactometers are standardized at 60° F., and, as the correct reading depends on the temperature of the milk, these lactometers contain thermometers. Knowing the lactometer reading at a certain temperature, the specific gravity can be easily ascertained. A simple rule is, add one-tenth to lactometer reading for each degree above 60° F., and to subtract one-tenth for each degree below 60° F. The corrected reading can be found at a glance by using the sliding milk rule, which will be described later.

QUEVENNE LACTOMETER

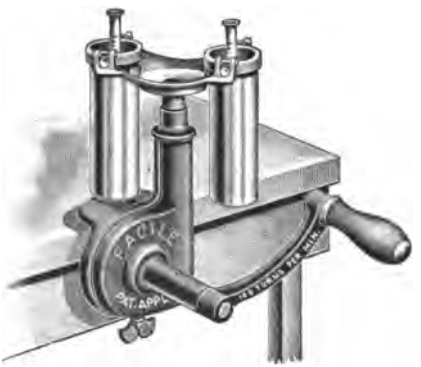
As the Quevenne lactometers on the market which contained thermometers were large and required a large quantity of milk, I had one made with a scale from 1,020 to 1,050 which only requires a little over half an ounce of milk to obtain a reading. This lactometer contains a thermometer so that the temperature of the milk can be ascertained at the same time.

The reading must be made with care, for important information is obtained from it. The specific gravity of mother's milk averages 1,030, and it is lowered by an increase in fat and raised by a high percentage of proteids. If the specific gravity of cow's milk is less than the normal (1,029 at 60° F.), one of two things may be assumed. Either the milk contains an unusual amount of cream, or the milk has been watered. If, on the other hand, the specific gravity is higher than normal, and the percentage of fat is low, it is fair to assume that the milk has been skimmed. The most important use of the lactometer is to determine the percentage of solids in the milk, and formulæ for this purpose will be given in another part of this paper.

The *percentage of fat* may be determined by many methods. We cannot at this time enter into a discussion of the relative merits of these methods. The principle of most of these is similar and consists of dissolving out the solids not fat of the milk by means of various chemicals, and the fat either measured as such in a narrow graduated tube or brought into solution with ether, gasoline, etc., and weighed on evaporation of the solvent. The test suggested by Professor Babcock, of Wisconsin, and known as the Babcock Test is the simplest and most accurate and has replaced most of the older methods. No expert chemical knowledge is required, and only one chemical is used. The

SEDIMENT TUBE

accuracy of this test is such that several states employ it for their official examinations, and it is in very general use throughout the United States. The manipulations are few and easily learned, while the apparatus is very cheap. The glassware consists of test bottles, a milk pipette and an acid measure. The test bottles have a capacity of about fifty cubic centimeters, and are made of heavy well-annealed glass. The neck of the test bottle on which the scale is sketched is three inches long and is graduated from one to five or one to ten per cent., and small divisions make possible the estimation of the fat into tenths of



CENTRIFUGE

one cubic centimeter with perfect ease. Special bottles are used for testing cream and skim milk.

The Babcock Tester is a centrifugal machine run by hand, steam or motor power. The machines on the market are all large and cumbersome, and are not practical for the physician's use. At my suggestion one of the large dairy supply houses, D. H. Burrell & Co., of Little Falls, N. Y., made a small hand centrifuge which is thoroughly practical, requires the regulation size Babcock test bottles, and takes up very little room in the physician's laboratory. In order to make this centrifuge of greater value to physicians, special sediment tubes are supplied so that the machine can be used for urinary and other work. These tubes have a detachable tip which is kept in place by cohesion of two ground glass surfaces. The sediment from a large

quantity of urine can be collected in the small cap, which is of great value in the detection of typhoid and tubercle bacilli. These Caps can be thoroughly cleansed and the sediment easily collected and removed for microscopical work.

MILK BOTTLE MILK BOTTLE

The Babcock test is made as follows: Seventeen and six-tenths cubic centimeters of the milk are drawn into the milk pipette and allowed to run into one of the test bottles. Seventeen and a half cubic centimeters of clean sulphuric acid with a specific gravity of 1.082 are measured in the acid measure and slowly run into the test bottle.

The milk and acid are thoroughly mixed in the test bottle by a rotary motion, placed in the centrifuge and whirled for four minutes. Boiling water is then added by means of the pipette until the lower part of the column of fat comes within the scale on the neck of the test bottle. A second whirling for one minute completes the separation of the fat. The test comes out clearer and nicer, and the fat is kept more limpid if boiling water is put in the cups which surround the bottles. The fat thus obtained should form a clear yellowish liquid quite distinctly separated from the acid solution. The bottles must be kept warm until the readings are made. The fat is measured from the lower line of demarcation, between the fat and the water, to the top of the meniscus of the upper line. The liability of error in reading is rendered less by using an ordinary pair of dividers. After the extent of the fat column is obtained, the lower point of the dividers is placed at the zero mark of the scale and the percentage of fat read at the upper point. The normal variations in both mother's and cow's milk are from three to five per cent.

The test requires an ordinary amount of care and a few cautions are necessary. The milk to be tested should be thoroughly stirred before the sample is taken. In order to balance the machine two tests must be made at a time, and if sufficient milk is obtainable it is a good plan to make a control test. The speed of the tester must be regular, and in order to obtain the clearest readings the milk and acid should be about 70° F. before mixing.

The mixing of the milk and acid must be done with care. When once begun the mixing should be continued until com-

pleted, for a partial or interrupted mixing will often cause some black material to separate with the fat and obscure the reading. This will also occur if the acid is too strong. The water added in order to bring the fat into the neck of the bottles should be soft, or else the carbonic acid gas liberated from hard water by the sulphuric acid produces a layer of foam on the fat and renders the reading more difficult.

The percentage of sugar is almost constant in both human and cow's milk, and it is not necessary to estimate the amount of sugar in an ordinary routine examination. Lactose will reduce copper salts, and the percentage of sugar present can be ascertained by the well-known Fehling quantitative test. Lactose will not ferment with yeast so the fermentation tubes cannot be employed. The average percentage of sugar in cow's milk is 4.5% and in mother's milk it is 6.5%.

The total solids of the milk can be determined from the data already procured, namely, the corrected specific gravity and the percentage of fat. Under the term "solids not fat" are included the nitrogenous matter, milk sugar, and mineral salts. These ingredients require complex chemical methods for their determination, but the ratio of these three constituents is rarely altered so that it is unnecessary, save in exceptional cases, to make a minute chemical examination.

A number of chemists have derived formulæ for the calculation of milk solids, and in recent years some of these have been greatly simplified. The Babcock formula has been found to be accurate and it is extremely simple. It consists of dividing the specific gravity of the milk by four and adding to this one-fifth of the per cent. of fat. For instance, the specific gravity of a specimen of milk is 1030.8 and the fat four per cent. One-fourth of the specific gravity is seven and seven-tenths and one-fifth of the percentage of fat is eight-tenths, so by the rule the total solids not fat would be eight and five-tenths. Adding the percentage of fat we get twelve and five-tenths as the total solids.

The formula constructed by Richmond is:

$$\text{Total solids} = .25 \text{ Spec. Grav.} + 1.2 \text{ Fat} + .14.$$

Using the same figures as in the Babcock calculation we would get

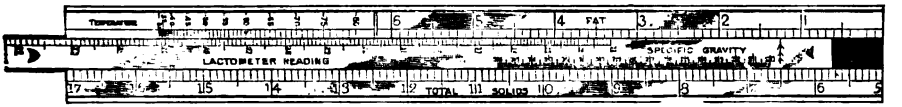
$$\text{Total solids} = .25\ (1030.8) + (1.2)\ 4 + .14.$$

or

$$\text{Total solids} = 12.6.$$

To further simplify this calculation Richmond has devised a sliding milk rule based on his formula. Both the corrected lactometer reading and the amount of total solids can be obtained at a glance, and it does away with the use of mathematics. This

rule has a movable slide in the center of its upper surface. On this slide are two scales, one representing the specific gravity, and the other the lactometer reading. On one side of the stationary portion a scale is placed, indicating the total solids; one per cent. of total solids equals one inch and is divided into tenths. On the upper portion of the other side the fat is shown



from one to six per cent. with each per cent. divided into tenths. On the lower portion of the same side a temperature scale is marked from 32° to 80° F. with an arrow opposite the 60° F. mark. The rule is operated as follows: The lactometer reading and temperature of the milk have been obtained. Set the rule, with the lactometer reading that has been observed, opposite the arrow at the 60° F. mark. For the true lactometer reading or specific gravity, notice the point on the movable part of the scale opposite the temperature at which the milk stood. For example, if the lactometer reading was thirty and the temperature of the milk 75° F., you would set the lactometer reading (30) on the 60° F. temperature line, and it would be seen that the 75° line corresponds with thirty-two on the lactometer scale, which is the correct lactometer reading.

The percentage of fat in the milk will have already been determined. The movable portion of the rule is adjusted so that the arrow points at the percentage of fat on the scale. With the scale thus adjusted, the line indicating the specific gravity or corrected lactometer reading is placed against the required percentage of total solids in the milk. Suppose, for instance, the fat is three and one-half per cent., the slide would be moved so that the arrow is opposite the three and one-half mark. The specific gravity was found to be thirty-two. Opposite thirty-two will be found twelve and four-tenths, which would be the required total solids in the milk.

The *amount of the proteids* can be obtained approximately by subtracting the percentage of fat, sugar and salts from the total solids. The percentage of sugar in mother's milk averages 6.5%, and rarely varies except in a small range. The average amount of the salts in mother's milk is two-tenths of one per cent. In cow's milk these figures are different. The sugar averages 4.5% and the salts seven-tenths of one per cent.

For example, the total solids in a specimen of cow's milk amount to 12.4% and the percentage of fat is three and one-half,

the solids not fat would be eight and nine-tenths. Subtract five and three-tenths, the sum of the sugar and salt percentage, and you would get three and six-tenths, the amount of proteids.

While this is a rough method, and throws all the error on the proteids, it gives us valuable information, which is reliable enough for ordinary modifications of milk.

It is sometimes desirable to ascertain if preservatives have been added to the milk. The most common one now in use is formaldehyde. If a violet ring is seen at the junction of the sulphuric acid and milk in the Babcock test bottle, formaldehyde may be present, although other conditions may cause it. This is the Hehner test and is said to be very delicate. The ring will not form if the percentage of formaldehyde is over five one-hundredths of one per cent. The phloroglucin test is more reliable. One gram of phloroglucin is dissolved in 100 cubic centimeters of distilled water. Ten cubic centimeters of the suspected milk are put in a test tube and five cubic centimeters of the phloroglucin solution added. One cubic centimeter of liquor potassæ (U. S. P.) is added and a pink color develops at once if formaldehyde is present.

The detection of other preservatives requires special skill and takes some time. Their presence, however, must be strongly suspected if the milk remains sweet to taste and smell when the amount of acidity registers three-tenths of one per cent. or over.

Discussion

Dr. Walter A. Scott, of Niagara Falls: I feel particularly interested in this subject of milk inasmuch as it was brought home to me last summer in an epidemic of diphtheria occurring in my city, which was traceable directly to the milk supply. During the latter part of August there occurred about 20 cases of diphtheria in the city of Niagara Falls, all the cases being on one milkman's route. On investigation it was found that this milkman received his milk from two farmers. One of them had had a case of malignant diphtheria in his family, and the disease had existed for two or three days before medical advice was sought. As soon as a physician was called the cows were taken to a neighbor's and the family had nothing whatever to do with the cows afterward. Exactly eight days after the breaking out of diphtheria in this family twenty cases of diphtheria developed in the city, and all within twenty-four hours. Investigation also showed that two or three weeks previously there had been one or two members in the farmer's family who had suffered from sore throat. There is at the present time an epidemic of scarlet fever existing in our city, which is largely confined to one milkman's route, but the investigation of this epidemic has not yet been completed. These cases show the importance of protecting the milk supply of a municipality, a subject which does not generally receive adequate attention.

Dr. E. E. SMITH, of New York: I was very much interested in the facts presented by Dr. Shaw as I recognize their value to the practising physician. It has occurred to me that in using standard solutions or in measuring off a quantity of milk, use should be made of the graduated pipette. A five or ten cubic centimeter pipette is a very valuable instrument in the hands of the physician. It can be easily washed out, and is more convenient to use than the burette. Another suggestion I would make is in the use of the instrument for taking the specific gravity. I see no reason why a properly corrected urinometer should not be used for taking the specific gravity of the milk just as it is used for urine. The method for estimating the proteids is not altogether sufficient. One important difference between mother's milk and cow's milk is in the relative percentage of the albumin and casein. In the cow the casein is far in excess of the albumin; in human milk the albumin should exceed the casein. When the amount of casein is increased in human milk infantile disorders are apt to occur.

XVIII. PERIDUODENAL ABSCESS SECONDARY TO ULCER OF THE DUODENUM

BY WILLIAM SEAMAN BAINBRIDGE, M. S., M. D.
NEW YORK

PERFORATIVE ulcer of the alimentary tract, involving the general peritoneal cavity, has of recent years deservedly received considerable attention. Although our knowledge is still far from complete, the time devoted to the subject has already yielded some very gratifying results. With increased data earlier diagnoses have in many instances been possible, and in some cases surgery has accomplished brilliant cures. Distinct advance has been made not only in typhoid and gastric perforations, but also in duodenal ulcer. Weir points out that the mortality in cases of perforation of the first portion of the small intestine was seventy to eighty per cent. But since 1898 operations for general peritonitis and suture of the perforation have reduced this, according to the length of time between the perforation and the operative interference, to thirty-three to sixty per cent. Moynihan, and Perkins and Wallace, diagnose only one case each of perforative ulcer out of fifty-one, and in eighteen of these the clinical picture was such a perfect copy of perforative appendicitis that the operation was performed with the firm belief that there was appendicular disease. Others have experienced like difficulties in reaching a diagnosis, and we must frankly admit that with our present knowledge, in the vast majority of cases,

the diagnosis must be general peritonitis, with the site of the perforation undetermined.

When we turn to the form of perforative ulcer of the duodenum, which does not involve the general cavity of the peritoneum, we find there is very scant information in medical literature. The subject is dealt with rather incidentally, and many important facts are either absent or stated too vaguely to be of real value. Although this disease is comparatively rare, yet it does occur often enough to be carefully studied, and kept in mind as a distinct possibility. Maydl, writing in 1894 upon subphrenic abscess, collected 179 cases, of which six were due to perforative ulcer of the duodenum. There are, including the author's case reported below, twenty-six on record. Twenty-two of these cases died without the diagnosis being made. In two an operation was performed and the diagnosis made at the time that the abdomen was opened. A cure resulted in one (Moynihan). In another, diagnosis was accurate, operation performed, but patient subsequently died from the disease (Lowson). In still another (see author's report below), diagnosis was made, but the condition of the patient prevented surgical interference.

The purpose of this paper is to give in condensed form an epitome of the literature on the subject, to stimulate a fuller record of cases which may occur from time to time, and to urge a more general recognition of the possibility of this disease being present in daily practice, where the symptoms apparently point to a far more common disease.

The following is now reported for the first time:

July 23d, 1899, I was called in consultation with Dr. Charles A. Ellis, of Sherman, New York. The patient was fifty-four years of age, a bachelor, merchant by occupation, and a native of New York State. His family history was negative, with the exception that his father died of pyloric cancer. His previous personal history was good, except for a mild attack of appendicitis thirteen months before, which lasted seven days, and recovery was apparently complete. From that time he seemed to be in good health, except occasionally experiencing slight tenderness in the right iliac fossa. He considered himself moderate in the use of both spirits and tobacco and was able to live a normal life of the active business man until the onset of the present illness.

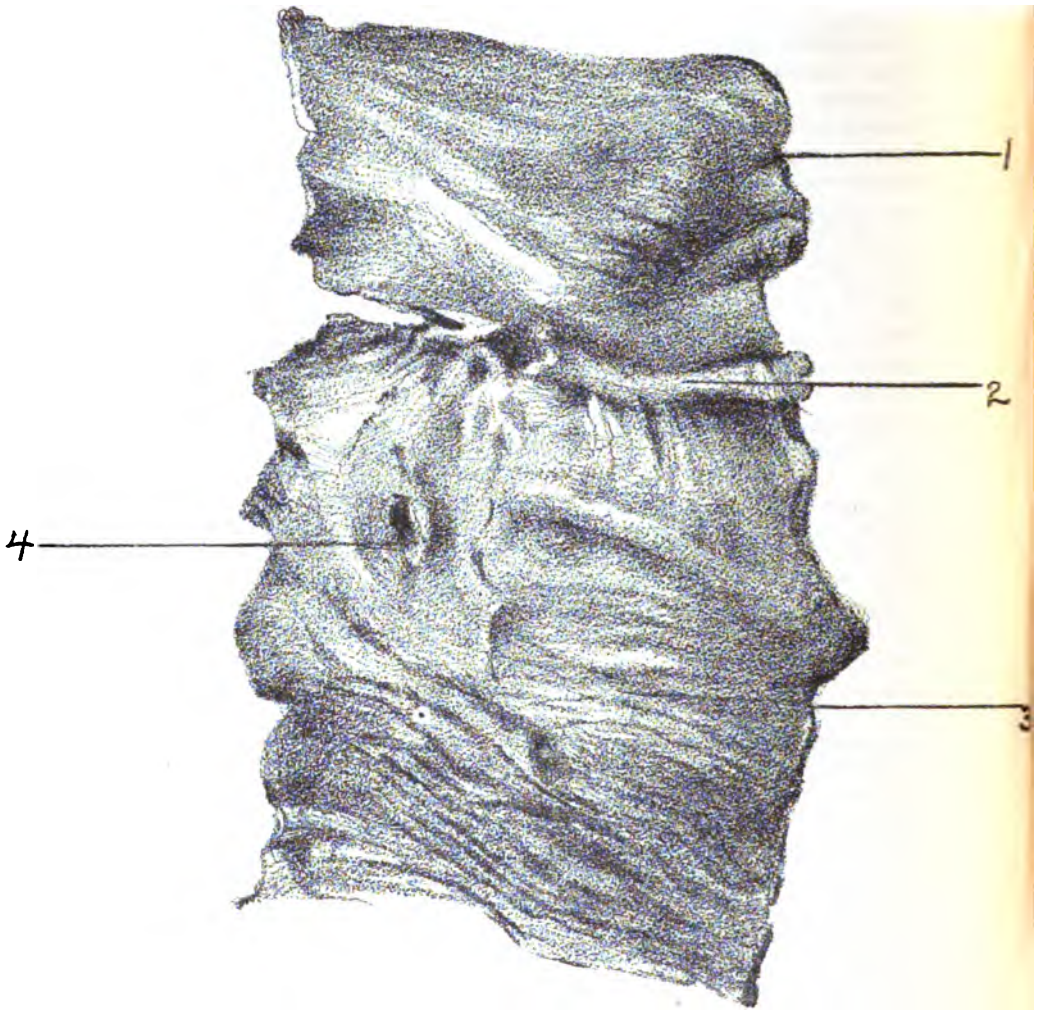
When his physician was first called on June 20th, the patient seemed to be suffering from an acute attack of gastritis. It came on suddenly, beginning with severe vomiting and intense abdominal pain, following immediately upon the ingestion of a glass of ginger ale. Some hours after the commencement of the symptoms, pain was still marked over the region of the stomach; there was tenderness on palpation, with considerable muscular rigidity over the ascending colon and the right half of the transverse colon.

The rigidity was more marked above the level of the umbilicus than below and the severe symptoms seemed to be more localized in the epigastric and right hypochrondriac regions. Considerable abdominal distension from intestinal gas was present. Temperature normal, pulse eighty-four, not very strong, respiration only slightly increased in rapidity and lessened in depth. Vomiting of watery fluid, at times of yellow color, and of everything taken, occurred occasionally for two days. With a diet of milk, the use of bismuth, and the employment of digestives, all symptoms gradually disappeared. At the end of six days from the onset the patient was able to walk about, seemingly well, although somewhat weak, and on this day made a short visit to his store. When he returned home, three small oranges were eaten at one sitting; a few hours later vomiting began which was frequently repeated during twenty-four hours. There was none of the very severe pain of the first attack, but epigastric discomfort and some abdominal rigidity. For two weeks rectal alimentation was employed, the stomach having perfect rest. On the fifteenth day food in small quantities was administered, and was retained by the stomach. By July 7th, fluid and soft diet with the use of liquid peptonoids were being constantly employed, yet the patient's strength seemed to diminish and emaciation progressed.

On July 23d, I found the patient markedly emaciated, somewhat cachectic, and the skin and conjunctivæ slightly icteric, temperature normal, pulse weak. The bowels, early in the attack constipated, were now loose. There was some tenderness and slight resistance in the region of the pylorus and the hepatic flexure of the colon. The liver was large and tender, its lower margin extending about three fingers below the free border of the ribs. Some resistance and pain on pressure over the appendix was manifest, and a small mass in this region could be apparently felt. The diagnosis was made, either of cancer in the neighborhood of the pylorus, possibly involving the liver, or more probably of perforating duodenal ulcer, with secondary abscess formation. The appendicitis was considered an old lesion with some acute congestion. The condition of the patient at the time precluded the possibility of any operative interference. Accordingly, it was decided to make every effort to secure temporary improvement with the hope that an exploratory operation might be undertaken. There was no difficulty in regulating the bowels, and apparently none in having a sufficient amount of food retained, but steady emaciation and loss of strength, with increased cachexia, continued. For a few days, by forced rectal feeding and with the use of predigested foods by mouth, a slight gain was made. But this was insufficient; again he gradually began to lose ground, and died of exhaustion the last of August, the eleventh week from the onset of the symptoms. Only twice was there any temperature, and that followed a slight chill, the second and sixth week of the illness. At times his temperature was subnormal.

On autopsy performed within a few hours after death I found the following conditions: Many adhesions about the pylorus and duodenum, as well as between stomach and posterior abdominal wall; a small ulcer in the duodenum, one quarter of an inch in diameter, with its edges everted.

The ulcer, as shown in the photograph, which was taken from an exact drawing of the specimen, was on the posterior wall an inch and a quarter from the pyloric valve. It communicated by a small opening with an irregular sac filled with almost a quart



1. Stomach. 2. Pylorus. 3. Duodenum. 4. Ulcer.

of greenish foetid pus. The abscess cavity was irregular and completely shut off from the general peritoneal cavity. It had involved the head of the pancreas, extending behind the stomach to the left, working downward underneath the transverse meso-

colon, and dissecting between the folds of the base of the mesentery. At the attachment of the mesentery, entirely extra-peritoneal, was at least a cupful of greenish pus. This communicated with the rest of the pus sac through a narrow channel, the walls of which were covered with necrotic tissue. A small arm of the abscess passed downward, external to the psoas muscle and posterior to the colon. This extended almost into the right iliac fossa, although the appendicular trouble was entirely shut off and independent. The appendix was enlarged, turned upon itself, and bound down by many adhesions. A small amount of turbid serum with fecal odor was seen between some of the adhesions. There was also recent general acute peritonitis, apparently of only a few hours' standing. The liver was large and the kidneys showed considerable paranchymatous nephritis.

A study of the literature on this subject reveals the fact that there are only about eleven cases reported sufficiently to be of scientific value in a comparative study of this disease, and in some of these important data have been omitted.

CASE I.—Sex, male; age, thirty-seven. Reported by Bouchard.

Previous personal history. Digestive disturbance for a month. Noticed a few hours after eating.

Onset of attack. Sudden sharp severe pain in epigastrium. Nausea and vomiting of food and bile.

Signs and symptoms. Vomiting, obstipation, continued severe pain in epigastrium and right hypochondrium, inability to obtain easy posture. Swollen abdomen.

Icterus. After a few days developed.

Temperature. Slight until late in the disease. Then high.

Diagnosis. Inflammation of gall passage. Pleurisy with effusion later when higher temperature.

Treatment. Patient was in hospital but no treatment mentioned.

Course of disease. Improvement. Left hospital. Returned in two weeks very weak and much emaciated. Died day after readmission. From onset of first severe symptoms to death about three weeks.

Autopsy. Fresh general peritonitis. Intestinal adhesions. Beneath diaphragm a large empty pus cavity. Perforation of the duodenum one and one-quarter inches in diameter near the pylorus.

CASE II.—Sex, male; age, thirty-three. Reported by Chauffard.

Previous personal history. Uncertain history of digestive disorders.

Onset of attack. Two days before admission to hospital sudden pain in right hypochondrium—obstipation. General weakness.

Signs and symptoms. Swollen abdomen. No vomiting for two days.

Icterus. Not mentioned.

Temperature. Not mentioned.

Diagnosis. Perforative peritonitis.

Treatment. Purgatives given without effect for two days.

Course of disease. Two days after admission sudden emesis with purgation. Death followed immediately.

Autopsy. Intestines distended with gas. Many firm adhesions. Large subphrenic abscess, connecting with the duodenum by a perforation one inch from the pylorus.

CASE III.—Sex, female; age, twenty three. Reported by Pfuhl.

Previous personal history. General weakness one month. Slight chills and fever. Pain in epigastrium and right hypochondrium. Anorexia.

Onset of attack. Sudden sharp shooting pain in right side of thorax behind increased by breathing.

Signs and Symptoms. Right Hypocondrium slighrly enlarged, painful on palpation. Several severe chills.

Icterus. Not mentioned.

Temperature. Temperature at onset 101.3°—gradually increased to 140°.

Diagnosis. Rt. Pleurisy with effusion. Several days later right pneumothorax with characteristic signs.

Treatment. Puncture in fifth interspace. Thick pus containing fat crystals.

Course of disease. Few days. So much pus evacuated that heart moved back into place and patient died in ten minutes from time of puncture.

Autopsy. Omentum adherent to abdominal wall and posterior to it a large pus cavity. Also a subphrenic abscess size of a child's head.

CASE IV.—Sex, male; age, thirty-three. Reported by Pusinelli.

Previous personal history. Not mentioned.

Onset of attack. Fell from a height of five feet. Abdomen injured. For two days kept at work. Pain developed. Vomiting bile stained masses after meals. Went to bed.

Signs and symptoms. Swelling in right hypochondrium. Painful vomiting continued after eating. Loss of weight. Signs of right pneumothorax.

Icterus. Present.

Temperature. 101°.

Diagnosis. Not given.

Treatment. Not given.

Course of disease. Third day air detected in swelling of right hypochondrium. Fifth day vomited dark gray masses—not lung tissue. Died on tenth day.

Autopsy. Diaphragm pushed up to level of third rib on right side adherent to base of lung. Subphrenic abscess cavity filled with air. Narrow channel connects this with second abscess size of an apple, which opens into duodenum just at the pyloric orifice.

CASE V.—Sex, female; age, forty-five. Reported by Nowak.

Previous personal history. Past two years slight nausea and occasional vomiting.

Onset of attack. Sudden sharp pain right side and dyspnœa.

Signs and symptoms. Cough. Pain in right axilla. Emaciation. Heart slightly displaced to left and signs of right lung compression. Abdomen tender. Liver dulness to three fingers below free border of ribs.

Icterus. Not mentioned.

Temperature. Not mentioned.

Diagnosis. Not given.

Treatment. Exploratory puncture between seventh and eighth ribs right side—negative.

Course of disease. Ten to twelve days after onset vomited and coughed up large quantities of foul pus. During next four months small quantities. Five days after puncture fat crystals found in sputum. Diarrhœa set in. Stools contained mucus and small clots of blood. One month later collapse and death.

Autopsy. Old cholelithiasis. Large right subphrenic abscess. Perforation of duodenum near pylorus. Perforation of a gastric ulcer. Recent dysentery.

CASE VI.—Sex, female; age, forty-eight. Reported by Perry & Shaw.

Previous personal history. None.

Onset of attack. While eating violent cramp-like pain in epigastrium.

Signs and symptoms. Diarrhœa at end of a week and continued. Kept on left side or back. Pain in any other position. Liver large. Indefinite tumor near umbilicus.

Icterus. None mentioned.

Temperature. Normal.

Diagnosis. None.

Treatment. None mentioned.

Course of disease. Continued diarrhœa,emaciation, weakness. One month after attack passed six onces of blood in stool for three days—then died.

Autopsy. Abscess between enlarged liver, anterior abdominal wall and intestines communicating with transverse colon by a small recent perforation and with the duodenum on its posterior wall by a large circular perforation with smooth edges. Large chronic gastric ulcers.

CASE VII.—Sex, female; age, fifty-four. Reported by Lennander.

Previous personal history. Well until five years ago—dyspeptic. Habitual constipation. One year before onset bloody stool with nausea and vomiting.

Onset of attack. Sudden severe pain in abdomen. Marked resistance felt in epigastrium.

Signs and symptoms. None stated.

Icterus. Nothing said.

Temperature. Not mentioned.

Diagnosis. Not mentioned.

Treatment. Two weeks after onset by an incision through the right rectus a number of small abscesses opened. Perforation found deeper, supposed to be in pylorus as gas and stomach contents were seen issuing from it. Sutured. Eight gall stones removed.

Course of disease. Not given.

Autopsy. Sutured perforation in duodenum to right of pylorus on anterior; another found on posterior wall.

CASE VIII.—Sex, male; age, nineteen. Reported by Fenwick.

Previous personal history. Heavy drinker. Vomiting began two days before accident.

Onset of attack. Fell twelve feet on to abdomen. Vomited everything taken. Pain in right inguinal region.

Signs and symptoms. Symptoms of shock. Constipation. Fulness in right inguinal region. Typhoid condition set in.

Icterus. Not mentioned.

Temperature. On admission 100.4°.

Diagnosis. Not mentioned.

Treatment. After readmission, Laparotomy. Large amount of pus from right groin evacuated. Later incision allowed much fecal matter to escape.

Course of disease. Gradually improved from typhoid condition and left hospital one month later. Readmitted in five weeks much emaciated. Found fluctuating mass in right groin. Bowels moved freely. Three or four months after second operation diarrhœa set in and in another month death from exhaustion.

Autopsy. Body greatly emaciated. Abscess channel from Poupart's ligament to under surface of liver communicating with perforation. Ulcer duodenum one-half inch in diameter five and a half inches from stomach.

CASE IX.—Sex, male; age, fifty-six. Reported by Pegram.

Previous personal history. Ten years ago had stomach trouble. Well since then.

Onset of attack. After hearty meal general dull pain in abdomen. Next day pain increased markedly. Localized in right hypochondrium.

Signs and symptoms. Constipation. Frequent emesis. Abdomen flat, rigid, very tender.

Icterus. Not mentioned.

Temperature. Not mentioned.

Diagnosis. Not mentioned.

Treatment. Laparotomy. Perforating ulcer anterior wall of duodenum near pylorus with subphrenic abscess. Pus evacuated. Ulcer walled off with gauze.

Course of disease. Thirty-six hours after operation patient died.

Autopsy. Ulcer and abscess as above. Also scar of healed ulcer posterior surface of duodenum. General peritonitis.

CASE X.—Sex, female; age, fifteen. Reported by Lowson.

Previous personal history. One month before onset pain in left side. Two weeks later chills, depression and cyanosis about lips and nose.

Onset of attack. Profound collapse came on a few days later with severe pain to left of umbilicus.

Signs and symptoms. Abdomen distended. Breathing shallow and frequent. Below liver to the right of the median line was a painful swelling of considerable size. Next day swelling disappeared after patient had vomited a large quantity of foul smelling matter.

Icterus. Not mentioned.

Temperature. Increased.

Diagnosis. Perforative duodenal ulcer.

Treatment. Laparotomy. Gas and pus evacuated from a subhepatic abscess connecting with a perforation of the duodenum. Suture of perforation and draining of abscess.

Course of disease. Three days after collapse, vomited bile. Continued for five days. Temperature increased, pulse rapid and feeble. Abdomen distended.

Autopsy. There is no information concerning the after history of this case; autopsy is not mentioned, but the deficiency of the article makes it almost certain that death occurred within a few days after the operation.

CASE XI.—Sex, female; age, twenty-nine. Reported by Moynihan.

Previous personal history. Several attacks of pain and vomiting during last five years. Blood at times appeared in the vomitus. Sense of discomfort one to five hours after meals.

Onset of attack. With one attack light temperature, emesis, tenderness in right hypochondrium and severe pain to right umbilicus.

Signs and symptoms. Not mentioned.

Icterus. Not mentioned.

Temperature. 104°.

Diagnosis.. Made during operation.

Treatment. Laparotomy eighth day. Adhesion about duodenum, gall bladder and pylorus. Abscess between duodenum and liver. Duodenal wall thickened with perforating ulcer at juncture of first and second portions. Tube and gauze drainage.

Course of disease. Recovery within one month.

CASE XII.—Reported by Bainbridge. See full report as given above.

Anatomy. The duodenum is practically a fixed organ, and, while apparently safe, because of its close relation to the spine and right kidney, is really in greater danger than other organs more superficially placed.

The site of the ulcer, in relation to the peritoneal covering of the duodenum, determines largely the subsequent history of the case. The first portion of the duodenum is usually about two inches long, and the peritoneum, except for a small extent of its posterior surface where the neck of the gall bladder is in close relation, entirely encircles it. The second or descending part is from three to four inches long and covered only on its anterior aspect by peritoneum. This anterior surface is divided into supra and infra-colic portions by the passage from side to side of the transverse meso-colon. Thus, when the perforation is in the first part of the duodenum the chances are largely in favor of its causing general peritonitis. When the ulcer perforates through the supra-colic surface of the descending portion, the intestinal contents will be discharged onto the upper surface of the meso-colon. It may then folow down the ascending colon toward the right iliac fossa, not infecting the general peritoneal cavity, being walled off for some time by newly-formed adhesions. Murphy and Neff have given this as a reason why perforative appendicitis is so often simulated. Perforation of the posterior wall, wherever uncovered by peritoneum, will give retro-peritoneal abscess. Maydl states that he has never found perforations leading to subphrenic abscess more than three centimeters from the

pylorus. Laspeyres gives the anterior wall of the upper horizontal portion as the most common site for perforation. Oppenheimer has collected data concerning five cases where the pancreas was involved. The perforation, when it involves the general peritoneum, is usually acute, but there have been a few instances where the process was more gradual, and adhesions shut off a localized purulent peritonitis.

Symptoms. Little can be added to what has been given above. The most frequent symptoms are vomiting, pain, tenderness and resistance in the epigastric and right hypochondriac regions, constipation or obstipation, sudden onset and shock. Many patients cannot lie on the right side, such a position causing more pain. Usually there is a previous history of gastric or intestinal disturbances. A number of cases have followed a direct injury to the abdomen or some severe physical strain.

Malæna or hæmatemesis are not common. They occur more frequently in ulcer than in abscess. Laspeyres says "thirty-three per cent. of duodenal perforations bleed and are very often fatal." Oppenheimer cites twenty-four such cases.

Temperature, as a rule, if present at all, is slight. Complications may develop and give rise to fever.

Complications which are mentioned as having occurred. General peritonitis, dry pleurisy and pleurisy with effusion, empyema, pneumothorax, pyo-pneumothorax, cholecystitis, cholelithiasis, ulcer or perforation of the stomach, infarction of various parts of the body, such as lung and duodenum, pancreatitis, perforation of the diaphragm and lung.

Diagnosis. Found more frequently in the male than in the female. Fenwick's proportion, as given for ulcer, of ten to one, is too high. Of the twenty-six cases of abscess recorded, fifteen are males, ten females, and one, the sex not stated. Occurs in young or old. Brooks reports a girl of twelve years, Pegram a man of fifty-six, and Perry and Shaw a woman of sixty.

The following must be specially thought of in connection with this disease—empyema, pleurisy with effusion, cancer of pylorus or liver, pyo-pneumothorax, hydatid cyst, hepatic abscess, intestinal obstruction, disease of the gall-bladder or its ducts, and appendicitis. Of all the conditions which periduodenal abscess simulates, next to general peritonitis, appendicitis is the most frequent. Mackenzie says he has "not met with an appendicitis case giving rise to general peritonitis where there was a history of repeated attacks of pain, but duodenal ulcer does give these attacks."

Blood examination for leucocytosis may be of assistance when cancer or some other non-purulent condition is to be excluded.

The diagnosis is always difficult. If symptoms have indicated the presence of ulcer (pain in stomach two to six hours after eating, point of tenderness to the right of the twelfth dorsal vertebra, vomiting specially hæmatemesis), and if sudden severe pain, marked increase of vomiting, or collapse appears, operation is imperative.

Exploratory laparotomy should be resorted to when abscess is probably present.

For those who may desire to review the literature on this subject farther than the limits of this paper will allow, a complete Bibliography is appended.

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DISCUSSION

Dr. LEWIS S. PILCHER, of Brooklyn: I desire to recall the case which I reported to this Society about the year 1890. Upon inquiry of the reader of the paper, I find this report has escaped his careful search because it was reported under a wrong heading, as a "Pseudo-Diverticulum of the Duodenum." Nevertheless, in all its essential pathological conditions it has been very accurately described in this paper. The case I refer to was that of a young man who was admitted to hospital suffering from fever and general symptoms of abdominal suppuration. There was present a tender tumor in the lower half of the abdomen to the right of the median line. The symptoms connected with this condition were quite recent and acute, and the necessity for operation seemed to be evident. The abdomen was opened and a tumor, inflammatory in character, was found behind the peritoneum at the brim of the pelvis on the right side. After surrounding the parts in the usual manner with gauze packing, the tumor was opened, and there escaped from it pus and undigested food. Infective peritonitis followed, and on the third day thereafter the patient died. On autopsy there was found in the transverse duodenum a loss of fully one-third of the posterior wall, making an opening about an inch and a half in diameter. This opened into a cavity behind the peritoneum, into which the contents of the duodenum were passing freely. As a consequence of this the progressive abscess formation had resulted, and the conditions already described.

Here was a man of nineteen in whom a long-standing ulceration of the posterior portion of the duodenum had been present for weeks and, perhaps, many months, and yet during the early part of this there had been no symptoms whatever. It was only in the later stages of development, on the approach of the tumor formation to the surface, that the man became noticeably sick. That such an ulceration of the duodenum can be present for a length of time without symptoms is a matter for consideration in connection with the pathological changes to which this portion of the bowel may be subject.

Dr. HENRY L. ELSNER, of Syracuse: There are several points in connection with this condition which need to be accentuated. These are really cases of subphrenic abscess. The subject has been thoroughly considered in a monograph published fifteen or twenty years ago, a most classical work on the subject. A point in connection with the case reported by Dr. Bainbridge which is of the utmost interest is, that, associated with this duodenal ulcer there was a parenchymatous nephritis. If one studies the statistics of duodenal ulcer it will be found that a large number of these ulcers are associated with nephritis. This is a surprising fact which has been lost sight of by the profession, and which has only been unearthed by a thorough study of these cases. The doctor says that in his case there was parenchymatous nephritis. Why this relation should exist it is difficult to say. Many explanations, purely theoretical, have been advanced. We have had in our hospital service and private practice during the past ten or fifteen years between ten and fifteen of these cases of duodenal ulcer. One of the leading physicians of Central New York was under treatment for two or three years for duodenal ulcer. He had both gastric and intestinal hemor-

rhages, and an associated parenchymatous nephritis. He was suddenly seized with all the symptoms described in the paper, and a diagnosis of subphrenic abscess and perforation of the duodenum from ulcer was made. The post-mortem examination confirmed this diagnosis. I do not think that in the majority of cases the diagnosis is difficult if the previous history of the patient is considered, and we find the presence of blood in the stools. The trouble is that in many of these cases there is a long interval between the reappearance of symptoms. For instance, in the case of the doctor to whom I have referred, intestinal hemorrhages occurred four or five years before his death. He appeared perfectly well, although there were always evidences of parenchymatous nephritis. While on his vacation at the Thousand Islands he was suddenly seized with all the symptoms of sudden perforation—collapse, subnormal temperature and localized peritonitis. The peritonitis was followed by all the symptoms of pus accumulation, but his uræmic condition and the associated myocardial changes argued against surgical interference, and the post-mortem proved that it would have been impossible to save his life, because of the far-reaching and dense adhesions. The pus had burrowed high up under the liver on the right side. The point which I wish to make in this discussion is, that duodenal ulcer is often associated with nephritis; that in these cases we first have for a long period the ordinary evidences of local disease in the duodenum; that when the perforation occurs there is usually a subnormal temperature with evidences of local peritonitis, and that, in addition, there is a positive leucocytosis.

Dr. BAINBRIDGE: I am very glad that Dr. Pilcher has given us the minute details of his interesting case which he had previously reported as under other title. Since the discussion with the doctor this morning, I have incorporated his case in the statistics as given in this paper. The patient treated by Dr. Elsner adds one to our list of those successfully diagnosed before death. Although the doctor is to be congratulated on making the diagnosis in this instance, I do not believe that statistics will bear out his assertion as to the ease of the diagnosis of either ulcer of the duodenum or periduodenal abscess secondary to ulcer of the duodenum. Dr. Elsner has referred to Maydl's "Classical Monograph," published in 1894. This monogroph is on Subphrenic Abscess and gives a list of 179 cases. Only 13 are intestinal, and of these only 6 caused by duodenal disease. (In my summary of cases, I selected three of these six.) This shows the comparatively small number of subphrenic abscesses due to duodenal disease; but cases like the one I have reported are not so rare, but they should be remembered as a possibility. With increased data, earlier diagnosis should be made.

XIX. HEPATIC BALLOTTEMENT IN PHYSICAL DIAGNOSIS

By A. L. BENEDICT, A. M., M. D.

BUFFALO

The size of the liver is, on the whole, best estimated by auscultatory percussion, as previously reported.* This method is seldom vitiated by intestinal tympany, as in the case of ordinary percussion. Palpation readily detects an enlarged or prolapsed liver, and usually, the lower edge of a normal or contracted liver. It is always well to check the results of one method, by applying another and the lower margin of the liver, as determined by auscultatory percussion, can usually be corroborated by palpation. Apparently, the liver is always felt lower than it is located by auscultatory percussion, on account of the thickness of the body wall, for which allowance should be made. The fluroscope usually shows the upper curve of the liver distinctly, but the lower margin is not usually so sharp, and with some tubes, especially those of great penetrating power, it may be impossible to locate the lower margin.

The writer would suggest ballottement or bimanual palpation† as an aid to palpation of the lower margin of the liver. The method is not applicable to obese patients nor to those with muscular or unyielding chests. In young patients, even up to the age of forty, the method is often successful. The physician should stand on the patient's right, use the edge of the right hand for palpating under the ribs, and press firmly, downward and backward, with the left hand, over the hepatic area. Meantime, the patient should assist the manœuvre by breathing deeply. It is often possible to feel the liver plainly by this method when ordinary palpation, without resistance from above, fails.

At first sight the liver seems much more movable during respiration, when viewed by X-rays, than when palpated or percussed. On more careful inspection, it will be noted that the liver is about twice as movable with reference to a fixed spot as it is to the ribs, which are used as landmarks in palpation and percussion and that, in tidal respiration, the respiratory movement, as referred to the ribs as landmarks, is insignificant.

Note.—The above method was clinically demonstrated through the kindness of Dr. N. G. Richmond, of Fredonia.

* Merritt H. Cash, Prize Essay, Trans. Med. Soc., State N. Y., 1895.

† Neither *ballottement* nor *bimanual palpation* is an entirely satisfactory term as the procedure enables the liver to be felt on its descent not the rebound, and as the organ is not actually palpated by both hands.

XX. THE CARE OF THE INSANE

By CHARLES G. WAGNER, M. D.
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INSANITY has always interested the world. Its history runs back through the ages to the earliest times and it has played a conspicuous part in human affairs in every country since man first came upon the earth. Other affections or diseases cause the loss or disuse of one or more members of the body, but insanity involves the unfortunate sufferer's whole being and changes his relations with all external things. It robs him of self-control; it takes away his responsibility before the law and, frequently, renders him a pitiable, helpless wreck, wholly dependent upon others for all his wants. It is an ailment that comes alike to the weak and to the strong; to the rich and to the poor, and brings the shadow of appalling disaster into the home where before was happiness and contentment. For the great majority of insane persons unable to care for themselves under the stress of their awful affliction the State is called upon to provide hospitals. These State institutions are, therefore, of vital interest to the public and especially to the medical profession at large.

The Nineteenth Century saw wonderful advances in the care of the insane throughout the world but the greatest strides were unquestionably made during the last quarter of that period. Scarcely more than a score of years ago our asylums were still prison-like structures, gloomy without and more gloomy within. With few exceptions, the wards were guiltless of ornamentation and the barred window, the locked door, the crib bed, the massive furniture fastened to the floor, and a dozen or more inhuman devices for mechanical restraint made up a picture well calculated to strike terror into the heart of the newcomer and to fill with despair the unhappy victim of prolonged incarceration.

Those of us who were familiar with the old airing courts—small enclosures with high board fences, commonly called by both patients and attendants the "bull-yard"—in which the insane were usually herded when out of doors for air and exercise can never forget their demoralizing influence. Attempts at suicide were common, indeed the whole atmosphere of asylum life was so depressing that even death was looked upon by many sufferers as a welcome change. When a new order of things looking toward the removal of bars from the windows, the destruc-

tion of the crib bed and the airing court, the abandonment of mechanical restraint and larger liberty for the individual patient was inaugurated the innovation was met with prejudice under the guise of conservatism, so strong as to imperil its success. Medical men who had grown grey in asylum work wrote and spoke against the new school as the immature efforts of tyros to teach their elders, and predictions were freely made that failure and calamity would inevitably result wherever the newer methods prevailed. That these false prophets were wrong is a matter of history. The new principles gained wider and wider adherence. The gospel of non-restraint was preached in season and out until everywhere it became the accepted belief and practice among progressive hospital men. Our State hospitals are to-day the product of a process of evolution. Old prejudices have not been laid aside in a day but gradually they have given way to better methods in both the medical and the administrative departments. Each institution instead of presenting the appearance of a prison is now more like a thriving town of from 1,000 to 3,000 people where the majority of the residents are everywhere busy and contented. Industry is the watchword and on all sides may be seen groups of men and women actively engaged in some congenial occupation. In the proper season planting the gardens and the farm, or harvesting the grain, spading the flower beds, mowing the lawns, repairing the roadways or assisting in the erection of new buildings are occupations often more beneficial to certain classes of the insane than any strictly medicinal treatment that has thus far been devised. Special shops are provided where a small army of patients find employment making a great variety of useful articles among which are brushes of almost every imaginable kind, ornamental floor mats, cane seats for chairs, boots, shoes and clothing for all the inmates of the institution. In the greenhouse patients assist in bedding the plants and arranging them on the benches; in the electric lighting station, the engine room, and the carpenter's shop patients are busy assisting the mechanics; in the kitchens they work with the cooks, and in the barns they help to care for the horses and cattle, everywhere cheerful and busy.

If the visitor enters the hospital and passes through the wards he is impressed by a general air of comfort. He notes the pictures and mural decorations, comfortable chairs and settees, rugs and carpets, pianos and books and he learns, usually to his surprise, that all of these refining influences help to convert the turbulent, profane, abusive man or woman into the quiet orderly self-helpful patient seen moving at will about the hospital premises throughout the day. He learns, perhaps for the first time,

that the insane man differs but little from his sane brother and that he needs but the treatment due to a fellow human being to lead him speedily in the way of conducting himself like one.

The largest possible individual liberty consistent with safety is allowed and thus many patients not strong enough to engage in systematic work or disinclined to employ themselves in this manner find health-giving exercise in roaming the country side gathering flowers and berries by the way in summer, and hickory nuts, chestnuts, and butternuts in the autumn. The question may be asked do not the patients to whom so much liberty is accorded run away from the hospital? On the contrary, those to whom this privilege is granted are the most contented people in the household. Remove the bars from the windows and the locks from the doors and you at once take away, to a great degree, the temptation to escape. It is the patient shut in on the locked ward that continually looks for an opportunity to run away. He who has the opportunity constantly before him rarely takes advantage of it. For a large part of the population of a State hospital, occupation is the key note of care and treatment after the acute stage of mental disturbance is past. When once the sufferer can be made to believe that he is still capable of doing some useful work in the world the germ of hope is implanted where before there was only despair.

But for the new cases suffering from acute attacks of melancholia or mania—and these constitute nearly all of the curable cases that come to the hospital for treatment—a different method of treatment is necessary. Such cases are examined on their arrival with the utmost care. The causes that lead to mental break down are minutely noted. A physician accompanies the sufferer to the reception ward where after a thorough physical examination of the heart, lungs, and other organs he is given a warm bath, placed in bed and a bowl of hot milk or gruel administered. If there be much excitement or restlessness a mild sedative is added. The special treatment then depends upon the form of the malady but in nearly all cases instructions are given to the nurse for the administration of hot milk or beef tea at short intervals, and to record on a chart provided for the purpose the pulse, respiration and temperature, and on other sheets the detailed symptomatology and treatment. If the patient will accept it, solid food, such as eggs, chicken, mutton or beef, is added to the diet and every effort is made to build up his strength and at the same time quiet and sooth any tendency to excitement. Every possible effort is made to persuade the sick person in the gentlest and most tactful manner to accept food voluntarily rather than by forced feeding, and often a reluctant patient who at

first refuses food will eat heartily when properly approached by a skillful nurse. If there be a strong tendency to both mental and motor activity we think it best that the patient be kept in bed for a few days and one or more nurses remain constantly with him to kindly but firmly keep him in a recumbent position. Such patients may often be given with great benefit ten or twelve eggs daily, especially in cases of melancholia. Besides the eggs large quantities of milk may be administered. The milk should be hot and if not well borne it should be diluted with lime water, or, if not rich enough cream should be added as the individual requirements may indicate.

Some times the sufferer from melancholia refuses to swallow any food whatever and then we must resort to the stomach tube to supply him with liquid nourishment in the most nutritious form obtainable. If a patient is extremely restless, constantly agitated, or inclined to violence, and does not sleep under the influence of mild hypnotics, we find the warm bath 98′ to 104′ F'. an exceedingly valuable sedative. Baths both hot and cold, the needle bath, shower and electric bath, the galvanic and faradic currents, and massage are valued adjuncts frequently brought into requisition.

Occasionally we have patients who do not yield to mild sedatives or even such hypnotics as the bromides, chloral, sulphonal, hyoscine, etc. Formerly such patients were confined in cells, put in straight jackets, muffs, belts and wristlets or mittens and otherwise harshly treated but now all such devices are regarded as barbarous and instead we employ trained nurses educated for the special duties they have to perform. These nurses through the exercise of kind and gentle discipline and ever watchful care have happily demonstrated that the modern humane methods are infinitely superior to the old way where force was the dominating factor of asylum treatment.

The treatment of the insane to-day briefly summed up may be said to be the provision of pleasant and sanitary surroundings, good nursing, proper medical attendance, suitable diet, entertainment, and congenial occupation.

I have gone somewhat into detail on this subject, although not exhaustively, for the reason that I wish to emphasize the fact that state hospitals are not merely places for the detention of insane persons, but are rather *hospitals* where the best possible care and treatment that can be devised should be provided for them. We hear the criticism that psychiatry is not keeping pace with general medical science. If we are lagging behind in this respect the cause is not difficult to find. Our state hospitals are in good physical condition; the food supplies are, in the main, sufficient and

wholesome, and the medical work, I think I may affirm, was never done better than at the present time. Much has been done, but the crying need of the hour is special hospital buildings for the care and treatment of the acute insane. Go to any of our large cities and visit the general hospitals for the sick and maimed. Observe how millions are lavished on these institutions to enable the medical staffs to do their work successfully and then visit our state hospitals and note how limited are the best of them in facilities for scientific work. Thus far but little has been done in the way of building construction other than the provision of wards for mere custodial care. There is not a state hospital in this great state adequately provided with facilities for the care and treatment of cases of acute insanity. In every institution we possess, the acute recoverable cases are crowded in on wards with chronic cases or assembled in close proximity with one another to their great detriment and it is an undeniable fact that many of these patients who ought to get well and resume their places as producers among their fellows fail to recover simply because suitable care and surroundings cannot be given them. The importance of this question of suitable hospital wards for recoverable cases cannot be overstated for, besides the incalculable boon that restored health is to the patient and his family, the loss to the state when such cases become incurable and a permanent tax upon its charity is simply enormous. The average cost of maintenance of an insane person is not far from $200 per annum and the duration of life from twelve to fifteen years; hence, if the lack of proper accommodations and care permits these curable cases to lapse into the hopeless degradation of chronic insanity the burden upon the public is heavy; indeed, it reaches far beyond the individual sufferer, for it not infrequently happens that when the family is deprived of a father's support the wife and children drift into the poorhouse to be maintained at public charge. It will, therefore, readily be appreciated that the construction of a building where each individual patient sent to a state hospital suffering from an attack of acute insanity shall be given the greatest possible chance for recovery must prove a paying investment to the state. Such a building should consist of small wards, each for ten or twelve patients, with separate individual rooms, well warmed, lighted and ventilated. The wards should be comfortably furnished and equipped with the best modern sanitary appliances including facilities for medicated baths of all kinds, electric treatment and massage; an operating room with the necessary outfit for aseptic surgery and glass enclosed verandas where the benefit of sunshine and fresh air might be fully available. A "special diet" kitchen should sup-

ply an abundance of nutritions food and a well ordered dining service should bring it to the patient hot and in palatable form. The best trained nurses in the hospital should be assigned to these wards and all the resources of the institution should be taxed to re-establish the physical and mental health of the patient. With such facilities as I have outlined and some special construction for the complete separation of those affected with tuberculosis from the other hospital inmates I believe our state hospitals would show better results in the way of recoveries than have ever been attained in institutions for the insane.

DISCUSSION.

Dr. FLOYD S. CREGO, of Buffalo: The work in the State Hospitals is excellent and, under the new régimé, it is probably equal to any institutional work of its class in the world. The great point is the need for hospitals, but not in connection with the hospitals for the insane, but as a part of city hospitals. All over Germany there are, in connection with every general hospital, wards for the care of the insane, and these are under the care of a competent alienist. This is what we most need in this country. In Bellevue Hospital there is a pavilion in which the patient is retained for four or five days. There should be a hospital division like that found in the Charité, and until it is secured these cases cannot receive proper treatment.

Dr. WAGNER: I think it is true that we need special hospitals in connection with city institutions. The latter should have such special wards; but that does not do away with the very great need of special hospital buildings at the hospitals for the insane. There are acute cases coming constantly, both from the country and the cities, to the State Hospitals for the insane, and these should have the best care that it is possible to give them.

XXI. ARGUMENTS IN FAVOR OF THE EXISTENCE OF A SEPARATE CENTRE FOR WRITING.

By HERMON C. GORDINIER, M. A., M. D.

TROY

No subject in neurology has attracted more attention or excited more discussion than agraphia. That is, whether or not the ability to write depends upon the integrity of a separate cortical centre, near to but absolutely distinct from that of Broca's area through which articulate speech is effected. Two diametrically opposed views are, at present, earnestly maintained. The first and older view is that a focal cortical centre for writing does not exist and that the inability to write is as much the result of a lesion of Broca's centre as is the inability to speak. A lesion of this centre, therefore always excites motor aphasia and agraphia. This is the one advocated by the earlier observers, Trousseau, Gairdner, Wernicke, Lichtheim and Hughlings Jackson, and now strenuously supported by Dejerine, Mirallie, and Wyllie, in Europe, and Collins in America. The second view, on the contrary, maintains that the act of writing is independent of the faculty of expression by articulate speech and its loss is due to the destruction of a cortical center, distinct from the motor speech or arm centres and its seat is probably located at the base of the second left frontal convolution. The supporters of this view believe that agraphia does occur as a distinct entity; that it may be unaccompanied by motor aphasia; and that when the two defects occur together both the motor speech and the writing centres of the association tracts connecting these centres are involved. This view is supported by Exner, Marce, Ogle, Charcot, Kussmaul, Oppenheim, Bastian, Beevor, Bramwell, and Collier, in Europe, and Eskeridge, Pershing, Mills, Frankel, and the author, in this country. In support of this view, I have the following propositions to offer with the discussion of each in their order.

First, That sufficient clinico-pathological data exists to show that motor aphasia and agraphia do not necessarily exist together, and that complete destruction of Broca's centre does not necessarily cause agraphia.

Second, that when motor aphasia and agraphia coexist, the

*Dr. Beevor states in a personal communication that his case is one of pure motor agraphia, with slight right-sided hemiplegia unaccompanied with aphasia, word blindness or word deafness.

lesion probably involves the bases of both the second and the third left frontal convolutions in the right handed or the association tracts connecting these centres with the visual or auditory word areas.

Third. Isolated motor agraphia, though exceedingly rare, does occur and depends upon a lesion of the base of the second left frontal gyrus in the right handed.

In connection with the first proposition, it is very interesting to introduce Trousseau's own words in reference to the subject. He says, "the inability to write is proportionate to the inability to speak. Aphasics write as badly as they speak, and those who do not speak at all, are absolutely incapable of writing." Marce, on the contrary, in 1856, stated that the centres of co-ordination for spoken and written words are different and separated from each other in position, since twice the patients could write freely when they were quite unable to speak. It is independent of the mobility of the hand, since when the hand has preserved all its powers, writing has been found to be impossible.

In support of the first proposition and to show the fallacy of Trousseau's reasoning, I will introduce abstracts of the following cases, one of the most typical of which was Trousseau's own case.

He tells of a robust young civil functionary, who, in an attack of unconsciousness, had entirely lost the power of speech. There was no paralysis of any parts of the body. He could execute all the movements of the tongue and lips with the greatest facility. He was able to discharge the duties of his office, notwithstanding the loss of speech, as he could transact all his business in writing. He delivered to Trousseau, an exhaustive history of his case which had been written out by himself. (Vol. xv. p. 756, Zeimssen).

Oppenheim reports another interesting case of a painter, who, for seventeen years following an apoplectic attack, was completely aphasic and could utter nothing save the words "yes" and "no," but could give a written account of his entire illness and could paint beautifully.

Idelson, Oppenheim's assistant, has recorded a case of a patient who had been cured by inunctions, from a left-sided hemiplegia, hemianopsia, and hemianæsthesia, who after a new attack, became completely aphasic and was not able to utter a single word or syllable, but was able to write perfectly during his entire illness.

Charcot records the case of a woman who was under his care at La Salpetriere. She had been seized with aphasia, but there existed no trace of paralysis, either of movement or of sensibility. In his patient aphasia was the sole morbid symptom, and atrophy of the left third frontal convolution was the only lesion revealed

by the autopsy. This was believed to be due to an old occlusion or narrowing of the external and inferior frontal branch of the middle cerebral artery.

Bramwell in the *Lancet,* May 22, 1897, describes a case in which acute softening, confined to Broca's convolution, produced complete motor aphasia with little or no agraphia. The absence of any marked agraphia, according to Bramwell, was forcibly illustrated by the fact that the patient, who was unable to answer questions in vocal speech, carried a note book about with him for the purpose of answering questions and carrying on a conversation in writing. Bramwell explicitly states that the case seemed totally opposed to the views to which the great majority of authorities on aphasia held, that destruction of Broca's convolution necessarily produces agraphia and that, as Gairdner long ago argued, the degree of agraphia is usually proportionate to the motor aphasia. It seems to support the view advocated by Bastian, which he also, independently, arrived at, *i. e.,* that the nervous impulses which set the motor writing centre into action do not necessarily pass through the motor speech centre in order to reach the writing centre and that destruction of the motor speech centre does not usually give rise to agraphia.

Prof. C. K. Mills has sent me the notes, with photographs, of the brain of a very interesting case supporting the theory of the dissociation of agraphia from motor aphasia. The lesion, as the photographs show, was limited to Broca's convolution.

Referring to his case, he says, "The main features of this case are as follows: The patient, after an apoplectic seizure, developed a peculiar form of aphasia, apparently without any preceeding paralysis or any other symptoms of focal lesion like visual blindness, hemianopsia, etc. When examined by me about nine years after the onset of the aphasia, he had almost complete inability to name persons and objects which he was able to recognize through all his special senses. He had also a marked form of paralexia or paranomia. When he attempted to read, although he understood what he was reading, he repeated an absurd formula of a few phrases. He had limited spontaneous speech, even using short sentences without concrete nouns correctly. He could write many single words correctly, sometimes misspelling however. He held his pen or pencil correctly and wrote such words as he did write with ease and firmness. He was not a motor agraphic.

Ogle, Bastian and Dickinson have, each, reported cases in which there was right hemiplegia and aphasia, though the patients could write with their left hand. Ogle's case came to autopsy and showed a patch of softening, involving the left third

frontal convolution and reaching downward and backward to the fissure Sylvius. The second frontal gyrus was normal.

I will refer, briefly, to a case recently under my care, of a boy who entered the Samaritan Hospital suffering from a depressed fracture of the frontal bone. He had several attacks of unilateral spasms of the face and complete aphasia. The depressed bone was elevated and several blood-clots removed, but the aphasia remained. He nodded assent or dissent when spoken to, recognized objects and seemed to understand, perfectly, printed or written language. There was no hemianopsia or paralysis of either arm. He was able to write his name the day following the operation and in a few days he wrote sentences, copied and wrote correctly from dictation.

Altogether the most remarkable case on record to disprove the views of Trousseau, Dejerine and their followers, is the one recorded by Guido Banti. It will be reported here in full. A right handed man, aged 36, who was able to read and write correctly, had a sudden apoplectic attack in 1877. Recovering consciousness in a few minutes, he was found to be suffering from right hemiplegia and loss of speech. The paralysis of the limbs disappeared the following night, although the inability to speak remained. The next day he was admitted to the hospital, and after a most careful examination his condition was found to be as follows: The mobility of the limbs on the right side had returned to their normal condition. There was no trace of paralysis of the face or tongue. The patient made ineffectual attempts to speak, but he could not articulate a single word, not even isolated syllables. He was much affected by his mutism and sought to make himself understood by gestures. When given the necessary articles and when asked to write his name, he did it immediately. Other questions were put to him to which he replied in writing. He wrote the details and description of his illness without hesitation. Various objects were shown to him, such as pieces of money, etc., and he answered, correctly, the name of each object, in writing. Then instead of giving him directions by word of mouth, I wrote them for him, in order to thoroughly convince myself that he was able to understand writing. He replied to these questions with perfect correctness. He wrote very rapidly and did not seem to hesitate to choose his words. He made no mistakes in syntax or orthography. He could understand, equally well, ordinary writing and print, and when one spoke to him, he grasped their meaning, at once, and never wished to have their questions repeated. I then wrote some simple words and asked him to repeat them aloud. This he could not do. I then pronounced some words and asked him to

repeat them after me. He watched the movements of my lips and tried very hard to obey, but he never succeeded in pronouncing a single word. The patient died in February, 1882, from an aneurism of the aorta. At autopsy, a patch of yellow softening was found, situated in the posterior third of the left third frontal convolution.

In summarizing the ten cases above narrated, we find that complete motor aphasia occurred in each without agraphia. In five of the cases no autopsies were had, but in each of these cases the clinical history was very plain, and there was no paralysis of the muscles of either hand. In five of the ten cases autopsies were had, namely, those of Charcot, Ogle, Banti, Bramwell and Mills. In Charcot's case the pathological lesion was an atrophy confined to the left third frontal gyrus, due to occlusion of the inferior branch of the sylvian artery. The only symptom was complete motor aphasia. In Ogle's case the third left frontal convolution was softened while the second left frontal convolution was found normal, and there was motor aphasia without agraphia. Banti, in his case, found, as the only lesion, an area of softening limited to the posterior third of Broca's convolution, involving only the cortex. This had caused in a right-handed person, perfectly able to read and write, motor aphasia without agraphia, there being no word deafness, alexia or hemianopsia. In the very carefully reported case of Bramwell's softening limited to Broca's convolution produced complete motor aphasia with little or no agraphia. In Mills' cases the lesion was confined to Broca's area and still there was no motor agraphia. Collins in his recent work on "The faculty of speeech," p. 177, makes the following statement that, "A patient with cortical motor aphasia is completely agraphic, never learns to write with his left hand, no matter how long he lives after the aphasia developes." This statement seems too sweeping, as the first part of the statement is disproven by the autopsies of the cases of Charcot, Banti and Bramwell, and the latter part, particularly, by the case of Ogle.

These cases, it seems to me, prove beyond a reasonable doubt that motor aphasia and agraphia are due to two distinct lesions, and may occur disassociated. They also prove that motor agraphia is not usually a symptom of a lesion limited exactly to the cortical area of Broca.

The cases reported by Broca, Tamburini and Marchi, together with the notes of an unpublished case, with autopsy, sent to me through the courtesy of Dr. John K. Mitchell, of Philadelphia, furnish very strong evidence in support of my second proposition. That is, when motor aphasia and agraphia coexist, the

lesion, probably, involves the bases of the second and third left frontal convolutions. In each case above referred to, the symptom-complex, motor aphasia with agraphia unaccompanied with hemianopsia, alexia or word deafness, was present. The cases of Broca and Mitchell are of such great importance in this connection, that I will give a short resume of each.

Broca's case a man, aged 84 years, came under his care, October 27, 1861, with the following history. In April, 1860, he had a slight apoplectic attack from which he recovered in a few days. No paralysis of the limbs occurred, but his speech was lost, save for four or five very simple words. His intelligence appeared normal. He understood all that was said to him and he made use of expressive gestures. He had been in this condition eighteen months prior to Broca's observation of him. To his questions he was able to reply only by signs accompanied by one or two syllables, pronounced hastily and with much effort. Broca asked him to write and, although his arm and hand were not paralyzed, he could not write a single letter. His sight and hearing were good. The tongue was protruded straight and was movable in all direction. The patient died Nov. 8, 1861. At the autopsy the lesion, an old hæmorrhage, was exactly limited to the posterior third of the second and third left frontal convolutions. This hæmorrhage was, doubtless, the cause of the apoplectic seizure which occurred eighteen months before, as the brain showed no other lesion.

Dr. Mitchell's case was a man, aged forty-four years, who was highly intelligent and well educated. His brother died of sarcoma of the brain. He had headaches for ten or twelve years which were attributed to eyestrain. These headaches have greatly increased for the past two or three years. In August 1899, he had an attack of difficulty in articulation, with numbness of both hands but no loss of power. The attack lasted ten minutes. Three weeks later, there occurred incoherent speech and violent twitchings, first of the right and then of the left side of the face. Later on, similar attacks appeared which in one or two instances, were followed by a semi-comatose state lasting for twenty-four hours. The headaches increased to an unbearable degree. In Feb. 1900 optic neuritis appeared. Up to this time a tumor seemed doubtful. In several attacks he had temporary loss of speech, followed by impairment of articulation, but with the consciousness of his loss of power of formulated speech, slight enfeeblement of the right arm was noted. His difficulty in writing was precisely similar in character to his difficulty in speaking.

At the operation a large endothelioma, measuring seven centimeters by eight centimeters, was removed. He died two hours

after. The upper third of the ascending frontal and the highest part of the superior frontal gyri, were not compressed by it. The lower posterior portion of the superior frontal and the whole of the posterior portions of the middle and inferior frontal convolutions, the lower two-thirds of the ascending frontal, except the extreme lowest part, were all compressed beneath it. The tumor did not extend beyond the fissure of Rolando and did not invade the brain tissue, but started apparently from the dura, pressed upon and caused thinning of the cortex and localized atrophy of the cerebrum. Autopsy and notes were made by Dr. W. G. Spiller.

The four cases above mentioned are the only ones, in literature, with which I am acquainted that present, clinically, motor agraphia and motor aphasia with the pathological lesion at the base of the second and third left frontal convolutions. In Broca's case there were no other symptoms present, save the inability to write and to speak and the lesion was limited exactly to the bases of the left median and inferior frontal convolution. In Dr. Mitchell's case, the lesion, a tumor springing from the dura, involved these same named gyri by constant pressure causing a thinning and atrophy of the cortex without involving the white matter. The symptoms present, apart from a very slight weakness of the right arm, were motor aphasia with agraphia, and the inability to write, corresponding exactly to the inability to speak. It would be impossible to explain away these cases on the supposition of a subcortical aphasia, the lesion being so placed as to interrupt the association tract connecting the visual word area with the motor speech or, more properly, motor writing centre, thus placing the agraphia in the category of a sensory type.

In none of the cases, was hemianopsia, word deafness, mind blindness, alexia, motor or sensory paralysis, present. These cases, therefore, support the theory of the separate existence of a writing centre located at the base of the second left frontal convolution.

Drs. Farnkel and Onuf, from a study of 104 cases of motor aphasia with autopsies, conclude that the function of writing is independent of Broca's area and the presence or absence of the act of writing is of no value in differentiating a subcortical from a cortical motor aphasia.

To verify my third proposition, that isolated motor agraphia though exceedingly rare does occur and depends upon a lesion at the base of the second left frontal convolution in the right handed, I direct your attention to the cases of Pitres, Charcot and Dutil, and Eskridge, together with my own case of a glioma at the base of a second left frontal convolution the only localizing

symptom present being complete motor agraphia uncombined with any form of aphasia, in a right handed person, without paralysis or hemianopsia.

In Pitre's case the intelligence was normal, there was paralysis of the right side without anæsthesia, which soon disappeared, there was no trace of word deafness or word blindness, nevertheless, the patient was incapable of writing spontaneously, or from dictation but could copy in fac-simile. This condition existed ten years. He had hemianopsia and was able to write with the left hand. Those who would give no credence to this case because of the presence of hemianopsia, explain the loss of writing on the supposition of a subcortical lesion interrupting the association tract between the left angular gyrus and the motor area of the left side. While any explanation of the case without post mortem evidence is purely speculative, the clinical symptoms certainly place it in the category of a partial motor agraphia. A subcortical lesion so placed as to produce hemianopsia and sensory agraphia, must, necessarily, have destroyed the optic radiations as well as the fibres connecting the angular gyrus with the motor zone, and because of its size, produced word blindness or alexia. The clinical record shows that word blindness did not exist and that the patient was able to copy in fac-simile. I have been able to find but six cases, with autopsies, of sensory agraphia, all of which were accompanied with mind blindness or alexia.

The case of Charcot and Dutil was that of a woman, who, after her first apoplectic attack, had no symptom except motor agraphia. Years later, a second attack left motor aphasia. After repeated attacks, there developed pseudo bulbar paralysis. There was no word blindness or deafness. At the autopsy, a focus of disease was found in the foot of the second left frontal convolution, another at the foot of the third left frontal convolution and three in the middle of the right hemisphere. This case seems perfectly simple if we could but prove that the oldest focus of softening was at the base of second left frontal convolution, as the clinical symptom, motor agraphia existing alone so long a time, would indicate. The presence of agraphia for years before motor aphasia occurred, certainly stamps the case as one of pure motor agraphia.

The case described by Eskeridge had no paralysis of motion or sensation, no sensory aphasia. His power of articulation was perfect except a little slow. He could talk, read printing or writing aloud or to himself and understood what he read. There was no difficulty in propositionizing and the movements of the lips and tongue were preserved except in uttering very long and hard words. Otherwise, it was impossible to discover any speech de-

fect. In writing he formed letters properly but transposed letters, words, phrases and sentences, so that it was quite impossible to read what he had written. Formerly he had been an expert penman. He was observed for two months and he tried to write a letter daily, consisting of a few lines. He spent two hours or more in writing or erasing words before he could finish the assigned task. At times he seemed to recognize his mistakes in spelling, and at other times he did not recognize them and was indifferent to them. In trying to correct his errors he would usually make a greater one. At the operation a cyst was found at the base of the second left frontal convolution, which had destroyed the cortex over an area of about half an inch in diameter and had extended into the white substance. After the operation, his writing improved very rapidly.

According to Dejerine and Collins, the ideal case to prove the existence of a centre whose function is to store graphic images, and whose destruction would cause agraphia, would be one in which there was inability to write spontaneously and from dictation, and in which no disturbance of motility, particularly no disturbance of the right upper extremity, no aphasia, no alexia, no verbal deafness, and no verbal blindness existed. Collins states "that if anything is needed to make the data of aphasia more reliable in the future and more utilizable as scientific evidence in support of, or in behalf of certain theories of the genesis of speech and localization, it is that the patient be studied methodically and carefully and when the case comes to autopsy, that the findings be recorded in scientific language, particularly the seat, extent and nature of the lesions. A study of subcortical lesions to be of much value must be done with the microscope."

Both conditions, I submit, are thoroughly fulfilled by my case, the notes of which are as follows:

The patient, Mrs. Lizzie W., an intelligent Irish woman, received her education in the public schools. Her age was thirty-seven, and she had been married fifteen years. Had had five children and one miscarriage at the third month. Her father died at the age of sixty years, of cancer of the face. Mother alive and in perfect health. Has a brother and a sister living and in good health. Has lost two sisters, one of Bright's disease, the other from consumption. The patient had always enjoyed excellent health up to the onset of the present illness, which began about Christmas, 1897, when she began to fail in strength and had several attacks of vertigo without falling, and unattended with nausea or vomiting. A short time afterward she developed severe frontal and occipital headaches, with an occasional attack of vomiting. The headaches continued with increasing severity until the fatal issue. In March, 1898, she noticed that her sight was failing, and that she could not read print without the use of glasses. At about the same time she began to complain of a numbness in the right

arm. At no time during her illness did she have a unilateral or general convulsion. Her bowels have been constipated. She has had perfect control over her bladder. Has menstruated regularly, and in a normal manner. Patient converses intelligently, makes no errors and her memory for past and recent events is good. At no time during illness has she had the slightest difficulty in giving expression to her thoughts by articulate speech.

Examination April 5, 1898. Patient five feet four inches in height; weight, 140 pounds. Physical examination of the chest and abdomen elicited nothing abnormal. Urine normal. Temperature 98.6 F., pulse 68. Head well formed and perfectly symmetrical. No evidence of previous injury, no exostosis. Percussion of scalp elicits no tenderness. Veins of forehead not prominent. Pupils moderate and equally dilated, and respond actively to light and accommodation. Wernicke's hemiopic pupillary phenomenon not present. No hemianopsia. Optic neuritis of both eyes, though more intense in right eye. Owing to paresis of the right external rectus muscle the right eyeball could not be rotated outward, but its movements upward, downward and inward were found normal. Diplopia existed. Excursion of left eyeball normal. Color vision normal. Drum membranes appeared normal. Hearing distance for watch in each ear was three feet. Movements of tongue normal. No fibrillation or atrophy. Sense of taste and smell normal. Soft palate and uvula move in a normal manner. No loss of sensation over buccal or pharyngeal mucous membranes. Movements of vocal cords normal. No difficulty in swallowing. No stiffness of neck muscles or those of spine. Both sides of face symmetrical. No evidence of facial paralysis. Patient is able to perform, in a normal manner, symmetrical bilateral movements. Grip of right hand is slightly weaker than that of left. Dynamometer: outer scale, right, fifty; left, seventy-five. No muscular atrophy anywhere discoverable. No stiffness of muscles. Fine and coarse movements of both hands and arms normal. Movements of lower extremities normal. Muscular power of both legs equal. The most careful examination of the skin of the entire body showed no sensory disturbances. No œdema or other vasomotor changes. The most careful reinforcement failed to elicit the presence of patellar tendon reflexes. No ataxia in upper or lower extremities. The sense of posture normal. Rhomberg's symptom absent. Gait perfectly normal. She has always been right-handed.

Speech. Patient recognizes and names correctly any object placed before her. She can recognize and accurately name, with her eyes closed, any familiar object when placed in her right hand, such as coins and the like. She understands perfectly what is said to her. She recognizes familiar hymns. She can repeat words after another and speaks voluntarily without the slightest hesitation, and always correctly. She can read correctly, with the aid of glasses, ordinary-sized print or writing. She recognizes numerals at once. The only difficulty present is the total inability to write. Although she understands, perfectly, written language and can read to herself or aloud, she cannot write, voluntarily, nor form, correctly, a single letter, and cannot write from dictation or copy. She holds the pen in a perfect manner, and performs movements with it as if to write. Her writing consisted, however, of nothing more than a series of united curves. She was

unable to write with her left hand, as she was not abi-dextrous. The fine and coarse muscular movements of the right hand are executed in a perfect manner, and there exists no paralysis of the muscles of the hand, forearm or arm. Before the present illness began she could write perfectly. She showed me some of her writing, which was excellent.

Without the slightest external manifestation of a syphililic infection, it was thought wise to give the patient the benefit of the doubt, and hence potassium iodide combined with mercury was administered for six weeks.

May 20. After six weeks continuous use of increasing doses of potassium iodide combined with mercury it was discontinued, there having been no amelioration of any symptom. No sensory or motor symptoms. No hemianopsia or alexia. Voluntary speech perfect. Cerebration, which was very active at my first examination (the patient answering the numerous questions put to her very quickly and correctly), was now quite slow. This slowness of speech was not in any sense aphasic in character, as all questions were answered correctly, only there was considerable deliberation before the answers came. Memory good. There is an absolute inability to write voluntarily from dictation or to copy. The patient holds the pen or pencil in perfect position, and the movements of the forearms and hands are normal. Has perfect control of sphincters. In no other particular was her condition different from that noted above, save that there is a tendency to fall or to stagger toward the right side. This ataxia did not exist at the date of the first examination. The patient has noticed this condition for about three weeks, and she thinks it is becoming gradually worse. It is an incoordination similar to that of cerebellar disease and is like that frequently observed in disease involving the prefrontal lobe.

This condition of complete agraphia continued throughout her illness, and was, with the exception of a gradual development of a slow cerebration and frontal ataxia (Bruns), the only localizing symptom present. At no subsequent time was there found the slightest evidence of motor or any form of sensory aphasia. She never had a partial or complete unilateral or general convulsion, never became paralyzed or developed any sensory disturbance. Hemianopsia or alexia were never present. The presence of agraphia without motor aphasia, with a gradually increasing slow cerebration and ataxia, coupled with double optic neuritis, with severe and continuous headaches, unaccompanied with alexia, led to the diagnosis of a lesion of the prefrontal lobe, probably a new growth, the situation of which I was led to believe, owing to the fact that the slow cerebration and incoordination which developed subsequently to the agraphia, was at the base of the second frontal convolution of the left side.

After six weeks of further suffering, there being no change in her symptoms, she consented to an exploratory operation, which was performed July 19, 1898, by Dr. E. D. Ferguson, of Troy, N. Y. The left Rolandic area was exposed after the method of Reid, under strict asepsis. The trephine opening was next made through the middle third of this area. On removing the button of bone the dura bulged into and through the opening, forming a very elastic swelling which pulsated feebly. On incising the dura there was an escape of a considerable amount of sub-arachnoid fluid, followed immediately by the formation of a troublesome hernia cerebri. The opening

was now enlarged forward by the Ronguer forceps, and a careful search was made for a lesion, but none was detected. The cortical area, exposed by trephine opening, appeared normal. While discussing the propriety of enlarging the opening still more forward, her condition became so alarming that it was decided to close the opening at once.

The vessels having been secured, the wound was closed with kangaroo-tendon, and a gauze drain was left in the most dependent part.

July 20, 1898. On awakening from her etherization, it was discovered that her right arm was paralyzed. She appeared very drowsy and stupid, and complained of great pain in the region of the wound. She was unable to move the right arm, and the movements of the right leg were impaired.

July 21, 1898. Patient rapidly passed into a condition of unconsciousness. No control over bladder. Pulse rapid and very feeble. Temperature 101 F. She died at 4 P. M.

Autopsy, July 22, 1898, at 10:30 A. M. Only the head permitted to be examined. Trephine opening, 4.5 x 4.75 centimeters. Elevation of hernia cerebri, above dura, two centimeters. Dura everywhere free. Superior longitudinal sinus contained a soft, recent thrombus. Vessels of pia injected. Left cerebral hemisphere. Marked hemorrhagic softening of the hernia cerebri and surrounding brain tissue. Pia not adherent save at location of the new growth, which was found occupying the foot of the second left frontal convolution, being distinctly separated from the arm area by the precentral sulcus, which sulcus, in this case, was unusually well defined. The growth was elevated about one-half centimeter above the surrounding cortex, was slightly irregular in outline, of a distinctly firm consistency, and oval in shape. Its longest cortical diameter was two centimeters. The pia was intimately adherent to it. On section, the tumor was seen to extend downward and inward in the centrum seme-ovale as far as the roof of the anterior cornu of the lateral ventricle, and forward to near the apex of the frontal lobe, involving almost exclusively the white matter of the second frontal gyrus. The left third or inferior frontal convolution through its whole extent was found (macroscopically) perfectly normal, as was its associated centrum ovale. The centrum ovale of the superior or first frontal convolution, was, toward its ventral part, infiltrated by the growth. The motor convolutions in their middle third formed a part of the hernia cerebri. This area was filled with minute capillary hæmorrhages, the result of the sudden relief of the intercranial pressure. The rest of the motor area and the underlying white matter was found normal. The supramarginal and angular gyri, with their associated white matter, was very carefully examined and found normal, as were the convolutions and white matter of the occipital and temporal lobes. The basal ganglia and the internal capsule showed no naked-eye changes. The convolutions, centrum ovale basal ganglia, and internal capsule of the right cerebral hemisphere, normal. The interior of the skull was found normal.

Histological examination. The histological examination of the tumor showed it to be a glioma of rapid growth. It was of a yellowish-gray color, rather firm in consistency, not very vascular, and was inseparably blended with the surrounding white matter. It presented no softened areas, and there was no softening of the white matter in advance of it. It was not

at all cystic in character. Sections of the tumor were placed, for four days, in Orth's fluid, and subsequently hardened in alcohols of increasing strength. Small cubes were embedded in celloidin, and stained with hæmatoxylin and eosis, von Giesson's picric acid fuchsin and hæmatoxylin, Weigert's myeline stain, and with Mallory's phosphomolybdic acid hæmatoxylin and phosphotungstic acid hæmatoxylin.

Microscopical examination of the left inferior or third frontal convolution, with the associated white matter, showed it to be normal.

The ventricles contained an excess of fluid. The brain stem, pons, cerebellum, medulla, and the cranial nerves showed no macroscopical changes.

This case seems to prove the existence of a separate and distinct cortical centre for writing, having the same relation to writing movements as the motor speech center has to speech movements. This centre is located at the base of the second left frontal convolution for the right handed, and probably in the same location in the right hemisphere, for the left handed. Destruction of this centre produces pure motor agraphia without aphasia or paralysis of either arm.

XXII. CONTRARY REACTION AND ITS BEARING ON PROGNOSIS.

By SMITH BAKER, M. D.

UTICA

With many people there is, primarily, but little besides aversion and repulsion to anything approaching the gross or forbidding. Birth, breeding and self-culture have all combined to produce a personality having a reactional certainty away from that which fixes attention on something less refined than ordinary. Painful pity and the more abstract sympathy keep such from joining those who promptly indulge themselves in the sensory satisfactions of exceptional shocks and tears.

There is an element, however, often atavistic, in almost everyone which more or less positively determines him unexpectedly to be or do just the opposite from that which is originally intended. With many, the very first opportunity to witness or hear about some brutal or vulgar affair, is greedily embraced. For a longer or shorter time the enchanting fascination holds in a relentless grip. Then comes the reaction, and they shrink away, in absolute wonder that even for a time they could have

been so possessed. Says Charles Dudley Warner, "It is difficult to explain the attraction which the uncanny and even the horrible have for some minds."

Similar pre-determinations hold in reference to reactions to every sort of anticipated experience. Thus, certain bicycle novices are prone to run into the obstacles they discern and would escape. Other people are bound to experience the most dreadful miseries their forward-looking reveals. Others still plunge headlong into the whirlpools of vice or bungling they are forever barring themselves against. All such people are eventually mastered by that which they would keep away from, and not do.

But it is in women that we probably find this *reaction by contraries* most frequent. Naturally, women do not like to be shocked or hurt; they are supposed to be correspondingly cautious and averse to putting themselves where they will be. Yet, in the matter of social departure, or of enduring life and its affairs, the very most striking fact is that they are always doing that which they did not intend. "You never can tell what a woman will do," is their own pronouncement of the fact. And the higher organized, the better bred, the more sensitive she is, the more definite the general uncertainty and the more certain the reaction by contraries seems to be. Especially do we see this in all the finer requirements for health attainment and health preservation, where the unreliability amounts almost to a passion.

If reaction by contraries be so frequent in health, so likewise is it the case in disease. Certain people will accept diagnosis, warning and instruction with thorough intelligence and purpose, and so give promise of speedy and satisfactory results. On the spot they resolve to follow directions implicitly. But the very fact of accurate knowledge and its consequent imperative seems to be sufficient to determine a directly contrary course. Very soon they find themselves undertaking everything directly against orders, and very little in obedience. "I don't know what's in me," they say. Contrary reaction seems to be their relentless tyrant.

A beautiful, refined, intelligent woman of thirty had suffered affliction and loss until her native neurasthenia had come to be enhanced by an acquired one of profoundest import. Under treatment she rapidly improved, and then necessarily had to stop. She was told that not for months would she be in shape to make important decisions, or to take important steps. To this she assented so intelligently and so comprehensively that all seemed safe and correspondingly promising. However, within

a month she had done everything contrary to advice and assent; and then, according to the rule, greatly wondered at her folly. Nor did the experience fortify her against a similar experience. Within a year an almost precisely similar course was pursued over again. I have sometimes thought that had I given her advice to do every opposite thing imaginable, then, for certain, would she have actually done just what I really advised. In such cases we have little reason to charge conscious disobedience or heedlessness. The patient is moved from within, and often against conscious judgment, to do the very thing counselled against, and unreservedly undertaken to do.

The same principle is manifest in the reaction to painful surgery. Dr. Lorenz, in a recent paper, regrets the remnant of aversion to the knife which still remains, on the ground that it often interferes with timely relief. Undoubtedly, such aversion obtains widely, and must forever do so, so long as human nature remains anything like what it now is. But, in an increasing number of instances, we must not forget the principle of contrary reaction. Averse as people undoubtedly are to being operated, repugnant as the glittering keenness of the knife may generally be, still, we note the most sensitive and primarily the most averse becoming fascinated by the possibility of operation; and this, so insidiously and yet so imperatively, that in spite of nature and experience, they become victims, and almost feel outraged if finally there proves to be no ground for operative interference. A highly organized, neurasthenic scholar, was positively told that his supposed appendicitis must be operated at once. On his way to the hospital he called on another for confirmatory diagnosis. The alarm was thoroughly proven to be false. He returned home, only half jubilant; and, at intervals during the some half-dozen years since then, has had to be braced against the recurring impulsion to the table, which, unassisted he seems unable to rid himself of, or resist. At such times he finds the very idea of an operation as vertiginously fascinating, as is the usual slough to the viciously inclined. All its imagery —the anæsthetic, the table, the surgeon, the nurses, the convalescence, the return home,—constitute a drama of such interest as to fairly impel him to do what sense and science forbid. In this he is greatly helped by the fact that surgery and its wonderful successes is now so much in evidence. He can hardly refrain from becoming one of its heroes, whether or no.

When we come to discuss the basis of contrary reaction, sometimes we strike hereditary predisposition, sometimes life-long cultivation in just such reactions to everything; oftenest, however, according to my observation, there is hystero-neurasthenia

of some degree or other. One thing is certain, there is a definite mental and nervous attitude—a sort of emotional erethism, or possibly, in cases, relaxation, which seems to be sufficient to determine results from beforehand—and this, for a certainty; which, if they could only be anticipated and estimated would be of inestimable value. We may call this impulsion-attitude temperamental; we may well say it is "subconscious." But all attempts to define the pre-determining factor, may and do finally mislead, in that they may include both too much and some things too emphatically. Always, we can say that the individuality is mixed, hypnoid and dependent. Generally, he or she is histrionic and auto-mimetic. The condition suggests hysteria surely enough. Always, neurasthenia may be premised.

Practically, we note that as a result of shock or stress, these people readily develop certain dissociations of physical and psychical conditions, and lose perspective in both directions. They read into actual disease multiple imaginings. Imagination itself often becomes freighted with dire aspects of physical suffering, and permanently so. Later, there follow certain deeply intrenched, persistent apprehensions, depressions, aboulias and pessimisms; or, in place of these, morbid fears or imperatives, sometimes miserably conscious, often vaguely so, yet, unconsciously determining everything about them and their lives. In the end also these people are sure to feel the anguish of disappointment with their own decisions, as well as with everything that has been done for them. The latter chiefly, because, their misery recurring, they must explain it in some way, and, laying it to the physician, nurse, or friend, or enemy is the line of least resistence for them to follow. Often, this state appears to be suggestively paranoid. Sometimes, it is quite this: and, while it may never develop into full-blown paranoia, the "crankism" seldom if ever weakens.

Now, the bearing of all this,—the physical reaction to contraries, and the mental as well, surely points to prognosis, both directly and influentially. Many a physician and surgeon has been puzzled to recognize outcomes quite antagonistic to the best-laid intentions and measures. In surgery the operator who can be wise and forehanded in this respect is indeed to be envied. Many a surgeon who has so confidently predicted relief from certain procedures has indeed been able to call his operation successful, but his client has forced a wide gulf between promise and fulfillment. The physician, also, who can properly distrust appearances and expectations founded on these may save himself shame and grief. Many a practitioner, where death has seemed absolutely to impend, has been chagrined to find his

predictions ignored by life itself. In fact, all who, in particular cases, intuit the contrary reaction with probability will be wisely governed in prognostic affirmation or otherwise. For it is useless to expect a straight response where nature has set the course directly the opposite.

Recognizing the fact of contrary reaction, we naturally try to see how we can profit by it. Here, so far as didactic intent and method are concerned, there must be failure. We do not yet know enough about the hidden springs of human conduct in any sense, to be able to furnish directions for its contraries. Empiricism itself has made very little progress in this. Only, as we sharply study these cases and collate results, will we, in time, reach conclusions that may be helpful. Meanwhile, the good will remain with the few who are on the lookout and allow themselves to be cautious where otherwise they might be bold.

Especially are these principles applicable, in cases of hoped-for relief of neurasthenic states by surgical procedures. In these cases there is most frequently an immediate relief that gratifies both operator and patient. But, later on, there is no assurance of permanent relief, whatever. A somewhat common observation is illustrated by a case referred to me by Dr. G.: Woman, forty-two, twice married, three children, menopause well advanced. Possessed for years of the notion that minor subinvolution was the cause of all her deep depressions and nervous breaks, she was advised that an operation would probably not bring the relief that someone had promised her it would. She got much better; then followed, under stress and hard work, a recurrence of former neurasthenic symptoms, which led her to a well-known and skillful gynecologist, who positively and frequently promised permanent relief by curettage and trachelloraphy. The operation was a fine one, and for the seven weeks she remained in the hospital she was comparatively free from trouble. Within a fortnight after returning home she broke again worse than before; and it took several months to lift her out of the slough of the old trouble, so aggravated by her new disappointment. Here, many years of neuro-psychical perversion was not cured by the operation; moreover, it was utter nonsense to lead her to the table under any such promise. For permanent relief of existing local conditions, the operation was sufficiently justifiable, if not absolutely demanded. Further than this, high authority in the gynecological world did not and could not carry her. Further acquaintance has revealed that she is constitutionally prone to the contrary reaction in many ways mentally, and to idiosyncratic results physically.

In trying to determine whether an operation will actually give

permanent relief to nervous symptoms, it seems to me that there should always be kept in mind:

1. Whether the neurasthenic condition is congenital or acquired, or both.

2. If acquired, then, how recent the acquisition.

3. To what kind of an environment must the patient necessarily return.

A case of congenital neurasthenia or hysteria that has been cultivated by wrong breeding from babyhood on is pretty generally hopeless. A case that has been acquired by faulty life promises relief mostly if there have been manifestations few in number, and these of comparatively recent date. In all cases, even the most favorable, so far as recovery and recurrence are concerned, it means ultimate failure when the patient must return to the environment in which the disease has developed. In all these cases it were better to keep the surgical and the neuro-psychical indications and predictions sharply differentiated. Relieve the physical conditions of course; but remember the possibility of contrary reaction and every allied source of disappointment, so far as neuro-psychical conditions are concerned.

XXIII. ERYTHROPLŒUM: A CLINICAL STUDY

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In 1876 there was brought to France from Africa the bark of a tree which was later recognized as the *Erythroplœum guineense*, one of the *Leguminosæ*. From the red color of the solutions made from the bark, the name is seen to be appropriate. This is also known as Gidu, Mancona, Doom, Ordeal, Casca, Saucy and Sassy bark. It occurs in flat or curved pieces of irregular size, about 6 mm. thick, covered externally with an uneven, warty and fissured corky layer, or deprived of the same, of a dull brown color. It is hard, brittle, of a fibrous texture internally, with pale yellowish spots, inodorous, of an astringent, somewhat bitter, acrid taste and when powdered excites sneezing. Its active principle is a colorless alkaloid, erythroplœine, which is soluble in water and alcohol. The only preparation of the bark is the ten per cent. tincture adopted by the British Pharmaceutical Conference, of which the dose is five to ten minims.

During the next five years the chemistry and physiological action of the drug were investigated in England by Brunton and Pye and in France by Gallois and Hardy and by Sée and Bochefontaine. With considerable uniformity in various reports its

physiological action may be stated to be as follows: The heart is slowed at first, later it becomes rapid. The ventricles contract regularly, but after large doses irregularly and stop in systole, while the auricles may continue to beat. This slowing is remarkable (1) from the regularity and energy of the systoles and (2) from the fact that during this slowing the uniform blood pressure is not altered by respiratory movements. The blood pressure rises because (1) of the increased energy of the heart and (2) of the contraction of the blood vessels; this condition persists until the heart becomes irregular when it falls. The respiratory movements are at first slower and fuller, but when the heart becomes feeble they are accelerated and during the period of irregular and feeble heart action they produce the so-called respiratory oscillations in blood pressure. Large doses produce vomiting and increased peristalsis. Moderate amounts increase diuresis; poisonous doses induce convulsions and later marked weakness of all muscles, and finally death.

The mode of action may be summed up as that of a muscle-poison acting upon the heart earlier because it receives a larger quantity of poisoned blood. Upon the vagus its action resembles digitalis. It is a vaso-constrictor by acting on the vessels themselves, the vaso-motor nerves or on some vaso-motor centre not contained in the medulla but probably in or around the vessels themselves. The respiration is influenced through the pulmonary branches of the vagus. It is sternutatory, because the powdered bark is an irritant to the nasal mucous membrane, causes vomiting by reason of its solutions possessing the same property, and is diuretic for the same reason and under the same conditions as is digitalis.

The alkaloid has been investigated by Lewin, Égasse, Liebreich, Theobald, Onodi, Alt and others. In a 1 to 2 *per mille* solution, it is locally anæsthetic. This anæsthetic condition may last several hours, but practically it is inferior to cocaine. It produces myosis, dims the cornea, causes headache, giddiness and even syncope. Its employment for this purpose has been abandoned.

The field of use for erythroplœum would seem to be limited to the heart and blood vessels when cardiac disease is or is not accompanied by dropsy. Its ability to slow the heart is rather greater than that of digitalis, but it is more decidedly a gastric irritant. Its vaso-constrictor properties are practically those of digitalis and ergot combined. It is rather less cumulative than is digitalis, using this term in the same sense that it is applied to digitalis.

My laboratory work, done upon this drug in Paris, in 1882, left a desire to investigate it clinically, for which no opportunity has

presented itself until recently. The following are the cases which illustrate its action:

I. Mr. B., June 6th, slight arterial (interstitial) nephritis, mitral regurgitation, dilated left heart, slight anasarca.

June 21, moderate diuresis was observed, cardiac first sound of better quality.

II. Mr. D., Mch. 14th, marked dilatation of left ventricle, mitral regurgitation, renal congeston, confined to bed.

April 25, considerable improvement in heart sounds, aortic second well defined, some diuresis.

III. Mr. J., Dec. 12, old fibro-myocarditis, dilatation, mitral obstruction and regurgitation, moderate congestion of kidneys, anasarca.

Dec. 17th, marked diuresis, murmurs distinct; anasarca lessened.

Dec. 25th, considerable improvement, pulse slow and hard.

IV. Mrs. T., June 1, aortic stenosis and mitral regurgitation, fibromyocarditis.

July 7th, but little improvement in subjective symptoms.

July 24th, feels much better, and heart-sounds distinct.

V. Mrs. G., July 9th, fatty heart, with some mitral regurgitation, œdema of ankles, attacks of syncope.

Aug. 3rd, cardiac first sound of lower pitch, no œdema, some diuresis.

VI. Mr. E., Dec. 16th, aortic stenosis, mitral stenosis, considerable regurgitation and dilatation. No anasarca.

Dec. 26th, condition somewhat improved, hands and feet warmer.

Jan. 16th, subjective symptoms markedly improved.

VII. Mrs. M., May 31, aortic stenosis and regurgitation, mitral regurgitation, with marked ventricular dilatation, syncope.

June 16, dilatation lessened, murmurs more distinct.

June 29, no attacks of syncope since last report, marked diuresis.

VIII. Miss E., Mch. 1st, mitral regurgitation, anæmia, renal and pulmonary congestion.

Mch. 20th, cough has disappeared, cardiac first sound louder.

Apl. 29, much improved, marked diuresis.

June 1st, anæmia better, no circulatory symptoms.

IX. Mrs. X., June 5, fatty heart, mitral regurgitation, dilatation, pulmonary congestion.

June 27, all pulmonary symptoms have disappeared.

July 11, still improving, heart-sounds distinct and dilatation now but slight.

The dose employed has generally been ten drops in a wineglass of water after each meal. The dates given are those upon which the sphygmographic tracings have been made.

The indications for the use of the remedy are those which Withering laid down in the eighteenth century for digitalis, namely, a rapid, low tension pulse with venous congestion. As compared with digitalis, it is decidedly more active in slowing the pulse; it also irritates the stomach more and, therefore, is more likely to cause vomiting, and possesses a more disagreeable taste. As a vaso-constrictor, it has greater effect than digitalis, —in fact as great as digitalis and ergot combined. It is less cumulative than digitalis. It seems to act rather upon the inhibitory than on the muscular system. As to constancy of effect in slowing the heart, strengthening the pulse and promoting diuresis, digitalis is rather more reliable. The use of this remedy then, should be confined to those cases of fairly competent heart with low vascular tension, in which it will show its effects more rapidly and markedly, and to those cases in which digitalis has lost its usefulness or has utterly failed.

XXIV. SOME POINTS PERTAINING TO THE THERAPEUTIC MANAGEMENT OF THE UREMIC STATE.

By HEINRICH STERN, Ph. D., M. D.
NEW YORK CITY

The diversity of opinion as regards various procedures and medicinal agents in the treatment of uremia can be ascribed only to insufficient consideration of the original affection on the basis of which it has developed.

Uremia, an arbitrary designation comprising a multitude of phenomena often unlike in character and intensity in various patients and in the same patients at different periods, is not the exclusive result of one and the same causative factor in every instance. True enough, all uremic manifestations depend upon the condition of the blood, but while this exhibits some characteristics, which are in a measure common to all types of uremia, the serum of every variety possesses some distinctive feature. Responsible for this is not only the different underlying renal condition, but also the specific material of the infectious disease in whose wake uremia may appear, and the decomposition products of the retained substances, the formation, quantity and specific state of which, are dependent upon duration of retention, oxidation and the physical condition of the serum.

In the large, white kidney, for instance, the passage of the

watery constituent of the blood is seriously interfered with; in contracted kidney there is nothing to prevent its outflow. While at the approach of and during the uremic condition extremes in blood composition may not exist any longer, the sera of uremia on the basis of chronic parenchymatous nephritis are differently constituted in some major points from those of uremia due to interstitial nephritis. The tendency to convulsive phenomena in the one, and to a comatose state in the other variety, may well be explained by the different constitution and physicoelectric behavior of the respective sera.

It is not my intention to dwell upon all discrepant features of the various uremic sera on this occasion; however, in order to demonstrate the necessity of a different and more rational management of the various types of the attack I cannot refrain from briefly alluding to the retention nitrogen, a factor most conspicuous on account of its presence in large amounts in the uremic serum due to interstitial nephritis and by its occurrence in noticeably smaller quantities in the serum of uremia following chronic parenchymatous nephritis or the mixed form of the affection.

Under retention nitrogen in the blood is understood all the nitrogen remainig in the liquid after complete removal of the albuminous substances. The average amount of retention nitrogen[1] in 100 cc. of normal blood serum is between 25 and 30 mgs.; in chronic parenchymatous nephritis without uremia the average quantity amounts to 40 mgs. and in the presence of uremia to about 62 mgs. in 100 cc. blood-serum; in chronic interstitial nephritis without uremia a mean amount of 82 mgs. and when uremia prevailed 130 mgs. retention nitrogen were found in the average; in the mixed type of nephritis without uremia in the mean 51 mgs., and with uremia 120 mgs. retention nitrogen in 100 cc. blood-serum were demonstrated. These figures show that in the blood-serum of chronic parenchymatous nephritis about 35% more retention nitrogen is contained than in the normal liquid; that in interstitial nephritis the retention nitrogen in the blood-serum occurs in twice the amount as in parenchymatous nephritis, and that in the intermediate type of the affection there is contained more nitrogen than in parenchymatous and considerably less than in interstitial nephritis. They further evince that the amount of retention nitrogen is increased in the uremic conditions following the three forms of nephritis and that in the uremic serum in parenchymatous nephritis the amount of retention nitrogen occurs in about half the quantity it is found in the serum of uremia arising as the consequence of chronic interstitial nephritis.

The enormous quantities of retention nitrogen in the serum of chronic interstitial nephritis undoubtedly stand in causative relationship to the natural termination of this disease—uremia. In chronic parenchymatous nephritis uremia is not only a much rarer but also a less grave complication. In about 75% of the cases of uremia due to chronic parenchymatous nephritis which have come under my observation I have seen recovery from the first attack. The comparative infrequency and mildness of the uremic seizures due to chronic parenchymatous nephritis are explained by the fact of the relatively small amount of retention nitrogen.[2]

The inconsistency of pursuing the same plan of treatment throughout in cases of uremia so unlike in their manifestations and arising from two (or more) so markedly discrepant substrata is obvious. In the following it is neither my purpose to draw the outlines of a complete management of the various uremic states nor do I wish to introduce a new remedial factor, but I want to pass in review some of the therapeutic agents we are familiar with at the present hour, and to point out their particular field of usefulness.

Sweating.—Promotion of the activity of the sudoriferous glands to enhance the excretion of retentia is indicated under certain restrictions in every instance of acute uremia. It has proved of special value when employed soon after the onset of the uremic phenomena. When the attack has persisted for some time, or when a pronounced comatose condition has supervened, sweating, as a rule, is of little or no avail. For reasons already stated, its application has a greater salutary and more lasting effect in uremia having chronic parenchymatous nephritis at its foundation; it is essential that it should bring about the desired result in a short time; too long continued, particularly after marked symptoms of improvement have made their appearance, its beneficial influence at first noticeable, may cease, or it may become a directly harmful agent. That is, by free perspiration considerable amounts of excrementitious substances may be removed from the serum and the watery accumulations; the quantity of water, however, by the medium of which the solids are excreted, is in proportion larger than it exists in the blood. This is evinced by the molecular concentration of the blood-serum, which is most always higher than that of sweat. Withdrawal of indefinite (because unmeasurable) large amounts of water from the serum, in other words, tends to increase still further its hyperosmotic condition and to decrease its conductivity.

If sweating is kept up by active measures, enteroclysis with a weak sodium chlorid solution, or hypodermoclysis with a hypo-

tonic solution of NaCl should be performed at certain intervals.

Venesection.—Abstraction of blood is a therapeutic measure of transitory value mostly, but serving a good purpose in many instances of uremia. The most lasting results of bleeding are obtained in cases following acute nephritic conditions, that is in such instances in which the patient is still sthenic and in which the blood has not as yet attained that degree of admixture and impoverishment which it exhibits during the chronic renal affections. Furthermore, uremic manifestations on the basis of chronic parenchymatous nephritis yield more readily to venesection than those which are due to contracted kidney. In children, abstraction of 100 cc. to 250 cc. of blood often effects immediate improvement; in adults not less than from 150 cc. to 250 cc. and if the condition of the patient warrants it, 300, 400, or even 500 cc. of blood should be withdrawn. When the uremic symptoms do not abide after venesection, the procedure may be repeated on one and the same day, provided the patient (abstracting the uremic state) is yet in a comparatively fair condition and the uremic manifestations are the result of an acute renal disturbance. In asthenic and far advanced cases, on the other hand, abstraction of blood is not only useless as a rule but often even injurious.

The frequent salutary results of venesection in the properly selected cases cannot be ascribed to the abstraction of a few hundred cubic centimeters of blood and the proportionate quantity of retained nitrogenous material; furthermore, withdrawal of blood does not in the least interfere with the molecular concentration of the remaining blood nor does it tend to increase the electric conductivity of the blood-serum. We possess no definite information that after bloodletting oxidation is enhanced, a process by which the retentia could be transformed into innocuous substances. Such a transformation—if it really occurred—could probably reduce the "virulence" of uremic blood in a chemicotoxic sense but it would not materially alter its physicotoxicity. The improvement afforded by venesection is likely due to nothing else but the relief of the vasoconstriction in the kidneys or in the central nervous system or in both these places.

Hypodermoclysis and Infusion.—Subcutaneous or intravenous introduction of a weak solution of sodium chlorid dilutes the blood, produces diuresis and compensates for the amount of blood lost by venesection. The effects are more or less transitory in character, but may endure sufficiently long to bridge over a critical phase in the uremic attack. Intravenous injection, of course, acts more rapidly and vigorously than when the solution

is introduced by hypodermoclysis, but its effects are correspondingly less lasting. A sodium chlorid solution which is isotonic to human blood contains 0.9% NaCl. It is the true physiologic salt solution and its intravenous injection after severe hemorrhage is followed by regeneration of the blood-plasma and restoration of the normal volume of blood. In uremia, however, it has been observed that an isotonic sodium chlorid solution does not act as efficiently as one which is hypotonic. For this reason a decinormal NaCl solution containing not quite 0.6% of the salt is usually employed in this condition. This is understood when we remember that the high osmotic tension of uremic serum is not so much due to its ionized as to its neutral molecules. To effect a more general ionization and conductivity water only is essential. We do not need additional electrolytes in the uremic serum which are introduced from without, but we should inject a solvent to facilitate dissociation of the electrolyte molecules existing in the blood. As plain water, however, may call forth too pronounced hypotonicity and as it is a poison to the erythrocytes producing their crenation, a weak solution of NaCl should be resorted to. I have used a 0.5% solution with the utmost benefit to the patient, especially in uremia due to chronic parenchymatous or acute nephritis. The comparatively small amount of nitrogenous retentia in the uremic serum in diffuse nephritis facilitates the reestablishment of its normal conductivity after subcutaneous or intravenous injection of the salt solution; on the other hand, in uremia having chronic interstitial nephritis at its foundation, when the retention nitrogen exists in enormous quantities, and when ion formation and ion movement are more or less paralyzed, not alone water but also goodly amounts of active molecules seem to be indicated. This, however, I wish to have understood as a hypothetic suggestion only, for it is an extraordinary fact that I cannot assign the improvement in a single case of uremia due to interstitial nephritis to the influence of the sodium chlorid solution alone, whether I administered it in hypotonic or isotonic solution, by hypodermoclysis or intravenously.

The minimum amount of the solution to be used should never be less than 125 cc. if injected intravenously, and 250 cc. when administered by hypodermoclysis. Smaller quantities, if at all, produce very limited and rapidly passing effects only. The maximum quantity of decinormal sodium chlorid solution which may be administered in the uremic state at one time should not exceed 750 cc. if employed subcutaneously, and not more than one-third of this if introduced by infusion. The procedure, however, may be repeated two, three, and more times during the 24

hours, especially when it directly precedes phlebotomy or if a considerable amount of blood has been abstracted.

As serum conductivity is enhanced by elevation of its temperature, more good will be accomplished if the saline solution when entering the organism exhibits a temperature above normal blood heat. If utilized by hypodermoclysis it should possess a temperature of from 40° to 41° C. at the moment it enters; injected by infusion it may safely be introduced into the vein at a temperature of from 45° to 48° C.

Administration of Morphin.—When after the employment of chloroform, vasoconstriction and convulsive phenomena do not diminish, small doses of morphin, although not exhibiting specific antidotal properties, may be resorted to. In uremia of acute nephritic states, more particularly, it may tend to bridge over a critical period, although it has never been proved to my satisfaction that the attacks would not have abated in case morphin had not been administered. Its eventual efficacy in controlling uremic convulsions may be due to its retarding the metabolic processes, thereby preventing further accumulation in the blood of katabolic substances, and to its augmentation of vascular tonicity. Convulsive manifestations arising on the basis of chronic parenchymatous nephritis also frequently decline in intensity or cease altogether after the injection of morphin; however, if we remember that about 75% of such cass (whether morphin was administered or not) recover from the first attack, we are forced to conclude that the influence of morphin cannot be a very potent one.

Uremia of chronic interstitial nephritis, characterized in almost every instance by a condition of tranquil coma, very rarely exhibits convulsive phenomena. In the absence of such manifestations there is no occasion at all to employ morphin, and the existence of a comatose state *a priori* precludes its administration.

While, therefore, morphin in small doses is inocuous in uremia of acute renal disease and chronic parenchymatous nephritis, and may possibly be a factor in effecting subsidence of convulsive symptoms, it is not indicated in uremia of chronic interstitial nephritis which presents an entirely different clinical picture, but being comagenous itself, may aggravate the existing comatose condition and hasten a fatal issue.

1 The figures relating to the amount of retention N. were obtained by Strauss: Die chronischen Nierenentzuendungen, etc., Berlin. 902.

2 Those further interested in this subject I refer to my article: A Contribution to the Pathogenesis of the Uremic State: the Probability of its Physicoelectric Substratum, *Medical Record*, January 24, 1903.

XXV. INCOMPLETE TRANSVERSE CONGENITAL OCCLUSION OF THE VAGINA, AND A THEORY AS TO ITS ORIGIN.

By SAMUEL M. BRICKNER, A.M., M.D.

NEW YORK

Incomplete transverse congenital occlusion of the vagina is a comparative rarity. In my service of upwards of 20,000 women at the Mount Sinai Hospital Dispensary, I have seen but three examples of it; and shortly afterward, another case came under my observation which illustrated the obstetric significance of the condition. In his exhaustive monograph,[1] Neugebauer described but fifty-seven cases of this kind out of 1,000 congenital and acquired atresias and stenoses of the vagina, and in this country very few cases have been recorded.

In the consideration of cases of congenital origin, we naturally exclude all those stenoses or narrowings of the vagina which have their basis in disease or injury. Stenosis of the vagina, for instance, may follow any of the acute infectious diseases, notably scarlatina, sepsis, cholera, smallpox and diphtheria, and is not infrequently seen as a sequel to injuries of the vagina, such as caustic douches. Cases have even been reported[2] where inflammation following coitus has resulted in a vaginal stenosis offering an obstacle to birth. But these instances, while of equal interest, are not relevant to our present purposes.

The four cases which form the basis of this paper were all examples of a transverse, incomplete occlusion of the vaginal lumen, and all of them were instances in which the occluding sæptum was present at birth; that is, in each case, the vagina was found occluded above the hymen by a transverse sæptum springing from the lateral walls, pierced by a minute perforation in its centre, except in the second case, in which the sæptum did not encircle the entire vagina.

Case I.—*Occlusio Vaginæ Transversalis Congenitalis Incompleta; Adhæsiones ad Cervicem.*—Bessie K., seen at the Good Samaritan Dispensary, June 28, 1902 (Protocol No. 143). Aged twenty-five years; married nine months. Never pregnant. Measles at fourteen. Her mother had five children; one other daughter, now seventeen, menstruates regularly. Patient has slight pain in back and abdomen.

Menstruation.—Regular every month since sixteenth year. Has never had any discharge before or since marriage. Last menses three weeks ago. Coitus not painful. Has never had any injury.

Chief Complaint.—Lack of sexual feeling. No pain on first coitus. Patient is not aware that she bled from rupture of hymen.

Examination.—Examining finger enters a blind cul-de-sac at the upper end of the vagina. No fornices or cervix are to be felt. In the centre of the cul-de-sac a transverse strand, like a scar, can be felt. This band

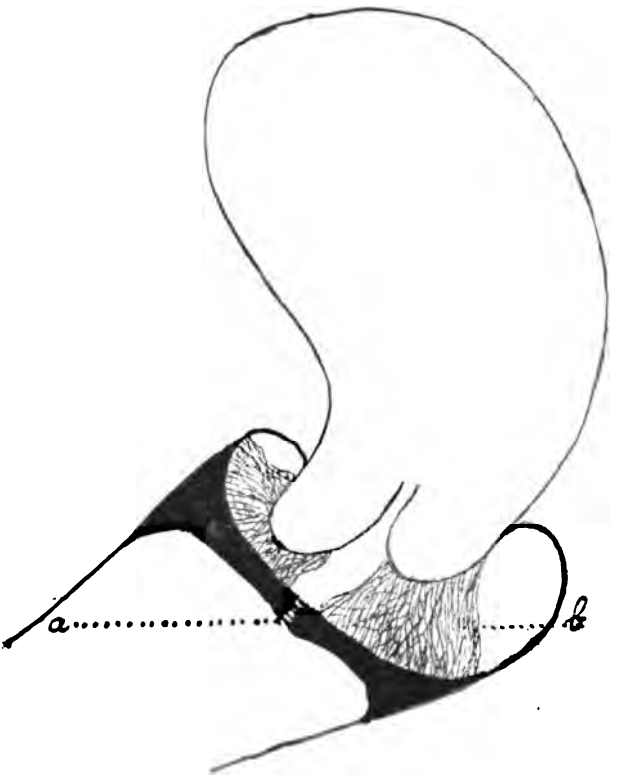

FIG. 1.—Case I. Incomplete congenital traverse occlusion of the vagina, becoming complete by a junction of the margins of the sæptum by granulation tissue. *a*, Granulation tissue joining edges of sæptum. *b*, Adhesions between sæptum and cervix. (Plates illustrating this article furnished through courtesy of the *New York Medical Journal*.)

is about one c. m. thick. With the speculum two granulation points can be seen in the transverse ridge and with great difficulty a silver probe can be forced through one of them. Through the opening thus made the point of a pair of scissors was introduced and the band was cut through. A very small cervix was then seen behind the band, and blood and inspissated mucus escaped. No bacteria were found in this fluid. The cul-de-sac was composed entirely of normal vaginal mucosa which on its posterior surface was adherent to the vaginal portion of the cervix.

Hymeneal remains are indistinctly seen at the vulva which is otherwise entirely normal. The vagina is short and rugæ are distinctly visible. The

uterus is two inches deep; the right ovary was felt, the left was not palpable.

The subsequent course of the case was one of improvement, the gauze which had been introduced at the first visit was repeatedly removed and the opening constantly enlarged. The adhesions between the cervix and the posterior wall of the sæptum were broken up by the fingers. The patient passed from observation when an operation was suggested.

CASE II.—*Occlusio Vaginæ Transversalis Congenitalis Incompleta.*—Bessie C.,[8] twenty-two years of age, born in Roumania, married eight months. Seen at the Mt. Sinai Hospital Dispensary, September 1, 1902 (Protocol No. 4,856b). First menses at thirteen years; menstruates about every six weeks for about five days. Has considerable pain after her period, lasting for five or six days. Her mother has had three children, all of whom are well. The patient is constipated.

Chief Complaint.—Abdominal discomfort.

Examination.—The myrtiform caruncles are distinct and are somewhat thicker than usual. The rugæ are present in the vagina and are well marked. At the junction of the upper and middle thirds of the vagina a tough,

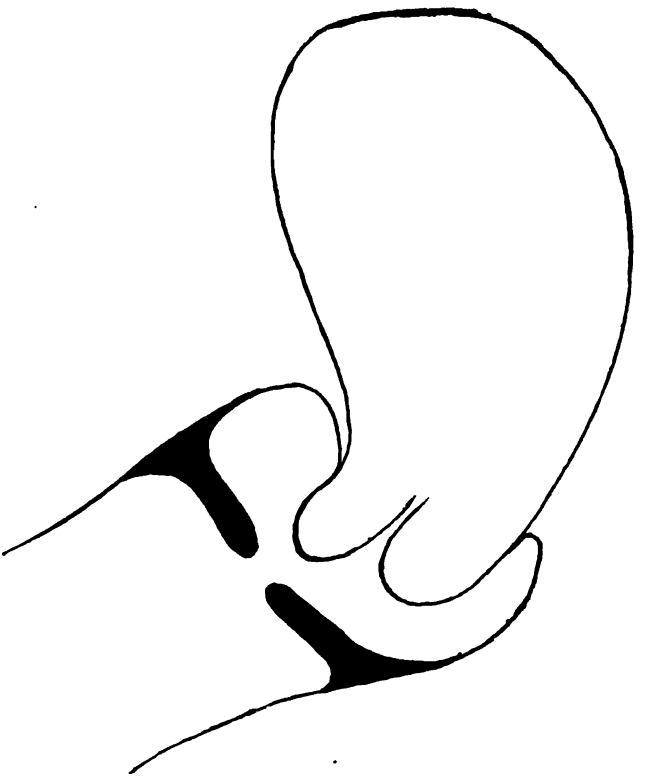

FIG. 2.—Case II. Incomplete congenital traverse occlusion of the vagina. This is the simplest form.

fibrous wall is met by the examining finger, encircling the entire vagina and being about a sixteenth of an inch thick. This sæptum is a little thicker on its left side than on its right. On the anterior vaginal wall, it approaches the cervix more nearly than elsewhere. The servix can be distinctly and easily felt through a central opening in the sæptum, about half an inch in diameter. The sæptum is not sensitive and lies free in the vagina. The cervix is small, conical, and easily admits the uterine sound. The uterus is anteflexed and is two and a half inches deep. The ovaries were not felt.

This patient also unfortunately passed from observation.

CASE III.—*Occlusio Vaginæ Transversalis Congenitalis Incompleta. Adhæsiones ad Cervicem.*—Dora W. (Protocol No. 5,140b, Mt. Sinai Hospital Dispensary, January 23, 1903), Roumania. Married three years. Never pregnant. First menses at sixteen years, then at intervals of eight months, six months, four months, and two months until it appeared regularly every month. Menses always painful on first day, but the patient has never been obliged to go to bed. The pain is in the back and abdomen. She has never had any acute infectious disease (scarlet fever, diphtheria, measles, smallpox, or cholera). Her mother has four daughters, all of whom are well and menstruate regularly.

FIG. 3.—Case III. Incomplete congenital traverse occlusion of the vagina, showing sæptum and central perforation.

The patient experienced pain on first intercourse and for six weeks subsequent to her marriage. Since then it has been painless, and there is no

complaint on that score from herself or her husband. Semen does not escape spontaneously from the vagina. The patient's chief complaint is pain in the back. She is also very constipated.

On examination, the vulva is found to be normal. The hymeneal remains are distinct, and are somewhat thick, showing that this organ was present and was of moderate thickness. The openings of the Bartholinian glands and of Skene's ducts are unusually well marked. A caruncle is seen in the urethra. On vaginal examination, the cervix cannot at once be felt, but it can be palpated behind a rather thick sæptum springing from the vaginal walls and completely surrounding the vagina. The cervix is very small, the uterus anteflexed. Ovaries and tubes could not be palpated. Examination by the speculum shows in front of the cervix a transverse sæptum with an opening directly in front of the external os, a quarter of an inch in diameter. Posteriorly, the sæptum is adherent so that the finger cannot enter the perforation. The sæptum is an eighth of an inch thick and is composed, apparently of normal vaginal mucosa. A striking fact is that the vagina contains no rugæ.

CASE IV.—*Occlusio Vaginæ Transversalis Congenitalis Incompleta; Dystocia.*—On September 27, 1902, I was called by a physician of this city to

FIG. 4.—Case III. With edges of perforation seperated, showing cervix lying immediately behind it.

see with him a parturient primipara, Mrs. E. F. The woman, I was told, had been in labor for eighteen hours, had strong, frequent pains, and at the time I was sent for, there was a bulging of the perinæum, yet the head could not be felt. Indistinctly, a hard body could be palpated through a membrane of some kind. The attending physician was much puzzled, having taken the membrane for unruptured amnion and having frequently, but in vain, attempted to perforate it. He requested my immediate attendance.

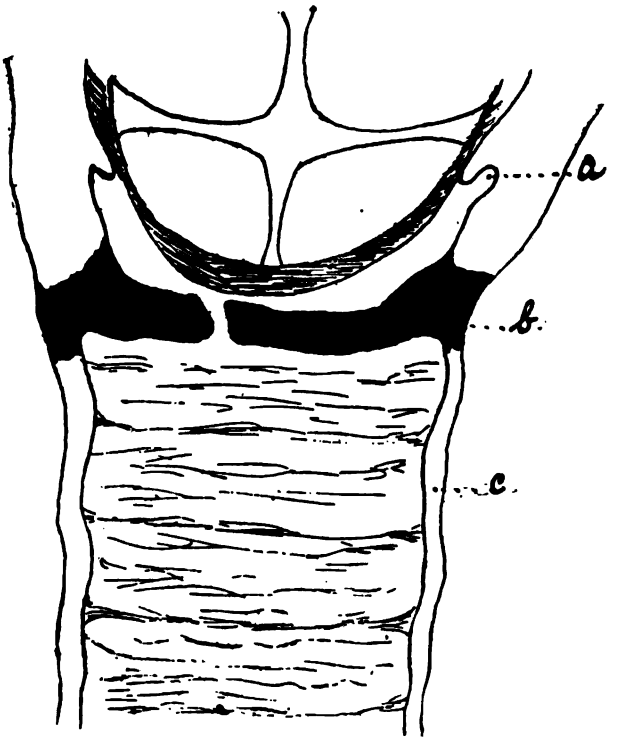

FIG. 5—Case IV. Incomplete congenial traverse occlusion of the vagina. Patient in second stage of labor. Traverse sæptum prevents descent of membranes and head. *a*, Internal os. *b*, Traverse sæptum. *c*, Vaginal wall.

Upon my arrival, a careful examination disclosed the nature of the case. A transverse sæptum of the vagina covered the descending head of the child, and, being elastic, it bulged down into the vagina with each succeeding pain. There was a minute opening to be felt in the centre of the sæptum. I decided to incise the sæptum rather than to dilate with the Champetier-de-Ribes's bags. This was done with a blunt-pointed scissors after making certain that the blades included only the sæptum. Four radiating incisions were made, extending not quite to the vaginal attachments of the sæptum. I took this precaution so that, if the descending head should tear the membrane, the lacerating force would be exerted upon it rather than upon the vaginal walls. There was a little insignificant bleed-

ing. The head lay immediately behind the sæptum, and in fifteen minutes the birth of a male child was successfully and easily accomplished, without injury to the perinæum. I was told, for I did not see the patient again, that the convalescence was uneventful.

After labor was completed, the vagina was tamponed to prevent the edges of the sæptum from cicatrizing with the vaginal walls and this was continued throughout the puerperium.

The patient's history before marriage was without special note. She

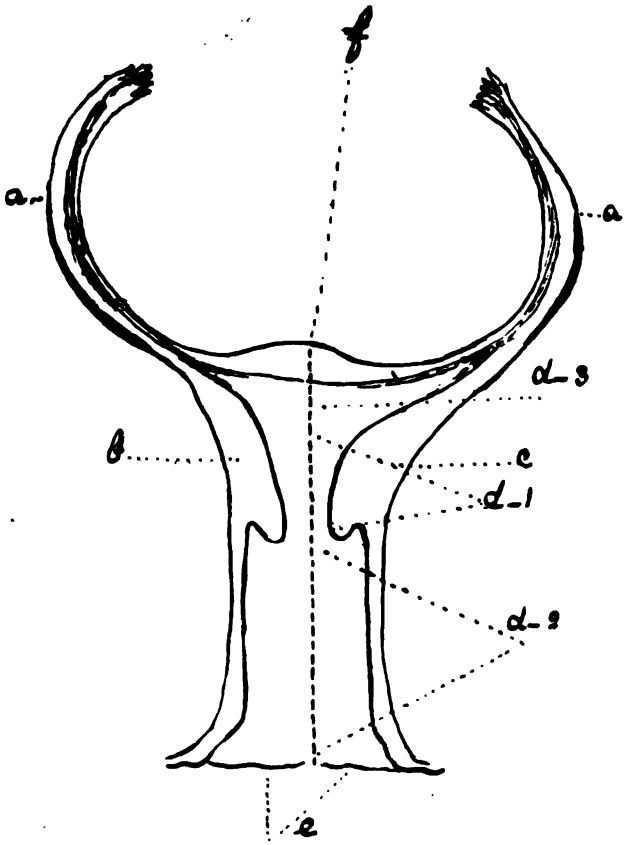

Fig. 6—Diagram showing fusion of Müllerian ducts to form the uterus and vagina, and their mode of disappearence to leave these organs hollow, *aa*, Fallopian tubes. *b*, From right Müllerian duct. *c*, From left Müllerian duct. *d*—1, First part of sæptum to disappear. *d*—2, Second part of sæptum to disappear. *d*—3, Third part of sæptum to disappear. *e*, Hymen formed at the end of the vagina by its junction with the urogenital sinus. [Modified from *Human Embryology and Morphology*, Arthur Keith, 1902.]

began to menstruate at fifteen, and was always regular, never having any pain. She was married in November, 1901, and menstruated for the last time, December 15, 1901. Twice during the early months of her pregnancy she had consulted her physician because of a lack of sexual feeling, and

she also told him of the complaint of her husband of his inability to secure complete *immissio penis*. No local examination was ever made, however, until the patient was in labor. Her pregnancy, I was told, was without any untoward event.

At my examination, the hymeneal vestigia were well marked and appeared like normal carunculæ myrtiformes. I mention this to exclude the consideration of the sæptum as a hymen situated high in the vagina.[4]

The Origin of Congenital Transverse Occlusions.—The formation of longitudinal sæpta of the vagina, as in cases of double vagina, is not difficult to understand when we remember the fusion of the Müllerian canals to form the genital tube, and their subsequent absorption at the points of contact. This absorption takes place along the entire length of the canal from the fundus of the uterus to the hymen. If that part of the amalgamated ducts which goes to form the vagina, fails of absorption, a longitudinal band will remain in the centre of the vagina, dividing it into two halves. It is necessary to remember in this connection that the primitive vagina is a solid body, and that by the absorption of Müller's ducts a hollowing out process takes place which leaves the uterus and vagina tubular. The hymen, too normally becomes perforated, although the only connection it has with the ducts of Müller is its fusion with the vagina at the urogenital sinus, and since the hymen arises at the urogenital sinus and fuses with the Müllerian canals at this site, it is impossible—as has been assumed especially by French writers—for a transverse vaginal sæptum to represent a supplementary hymen.

From these very brief data, it is plain that any kind of abnormity in the lumen of the vagina, from an imperforate hymen to a complete atresia of the vagina, can be traced back to a failure in the normal conduct of Müller's ducts. But the facts given do not account for transverse sæpta. Several authors have given attention to the subject, and their theories shall be considered before stating my own views.

Delaunay[5] explained the presence of these sæpta by assuming that the vagina developed in three sections, as though one were superimposed upon the other. If the middle portion did not undergo absorption, a transverse sæptum remained at the junction of the upper and middle thirds of the vagina. If the lower third failed to follow the normal course, a transverse sæptum would mark this fact at the junction of the lower and middle thirds. While this theory accounts for the simple sæpta, it leaves unexplained the presence of multiple sæpta. Schröder[6] and Breisky[7] believed that transverse sæpta represented inflammatory processes during fœtal life. Of this there is not only no confirmatory evidence, but it is difficult to understand how an in-

flammation can attack a solid epithelial mass without leaving more gross lesions than are shown by a simple, non-adherent, free lying band in the vagina.

Verchère[8] asserts that the transverse sæpta are persistent remnants of the amalgamation of the Müllerian ducts. He takes the well known embryological fact that the vagina constantly increases in length during embryonic life as the basis of the formation of a transverse sæptum. The two lateral openings which he describes are supposed to represent the lumina of the original *Anlage* of the Müllerian canals. This theory is ingenious, but it fails also to account for the presence of two or more sæpta, and most frequently, besides, the opening in the sæptum is central, and is single.

Magendi-Husté[9] has elaborated a theory which is more in consonance with embryological and clinical facts. If the primary solid epithelial mass which forms the vagina fails to be hollowed out from top to bottom, as is usually the case, and if at the point of this failure of canalization there is an additional defect of absorption, a transverse sæptum must result. This will be perforated or imperforate, depending upon the extent and nature of the hollowing out process.

These theories are many of them ingenious, and some of them account for some of the clinical and embryological facts; but they fail to take into account the complications and abnormities of this anatomical peculiarity.

Embryonal Defects.—My own conclusions from a study of the cases may be considered twofold: first, that transverse vaginal sæpta are due to an embryonic fault, and secondly, that these embryonal defects are due to a reversion to type, that is, a "return to an ancestral type." Dr. George S. Huntington, professor of anatomy in the College of Physicians and Surgeons, of New York, with whom I have discussed this topic, is inclined to the former view, and his opinion is certainly of weight; yet, while I lay the greater stress upon the theory of faulty development in the embryo, it has seemed to me that the belief in reversion cannot wholly be disregarded.

Let us now consider the evidence under the head of embryonal defects, without, however, entering more deeply into embryology than is essential for the thorough comprehension of the subject. The Wolffian bodies, later to develop into the most important of the excretory organs of the body, arise from the epiblast, or ectoderm, according to Kollmann, whose view is now universally accepted. In their immediate neighborhood, and springing from an invagination of the mesothelium of the cœlome, or body cavity, the Müllerian ducts soon appear. With these elements we are

mostly concerned. The ducts of Müller extend downward from their point of origin just beneath the diaphragm to join the urogenital sinus. This is preceded, however, by the development upon the Wolffian ridge, the differentiated portion of the Wolffian body, by a change in the character of the mesothelium upon its upper surface by which it becomes columnar in form, the same type of epithelium which is found lining the uterus and upper part of the cervix. From this time on, the Müllerian ducts of the two sides grow downward and inward by a proliferation of their cells until they finally fuse together in the centre (the third month) and also with the Wolffian ducts as these two reach the pelvis where they conjointly form the genital cord. From the Müllerian portions are developed Fallopian tubes, uterus, and vagina, while the Wolffian ducts supply to the lower third of the vagina, at least, its lining epithelium. In briefest outline, this is the story of the origin of the female genitals.

Let us now assume that during the formation of the genital cord, fusion between one portion of the ducts of Müller and one portion of the Wolffian duct becomes more intimate than elsewhere, that an invagination (*Einstülpung*) of a portion of the

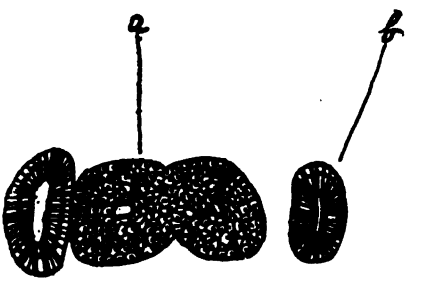

FIG. 7.—Cross section of genital cord. *a*, Müller's ducts. *b*, Wolffian duct. [From Herwig, after Tourneux and Lègay.]

Wolffian duct into the fused ducts of Müller takes place. Inclusions of this character are not unknown, especially in the genital region, and a glance at Figs. 7 and 8 will show how, under favorable circumstances, this inclusion or invagination may occur. But a single proliferating cell from one of the Wolffian ducts will be necessary to account for the peculiarity of subsequent development. Granted that this is the case, the subsequent course of Müller's duct will be modified in the following manner: It remains tubular in all places except where the inclusion has occurred, and this can only be in the vagina, because the fusion of the two sets of ducts into the genital cord takes place below the level of the uterus. The invaginated epithelium from the Wolffian duct continues in its course, while the fused Müllerian

ducts later become solid epithelial masses, only to become finally tubular by absorption of the epithlial masses and their own joined surfaces. But the sæptum formed by the invagination from the Wolffian, or epiblastic, epithelium, is not susceptible to the same influences as the mesoblastic epithelium, which becomes absorbed to furnish tubes, uterus, and vagina, and remains as a permanent

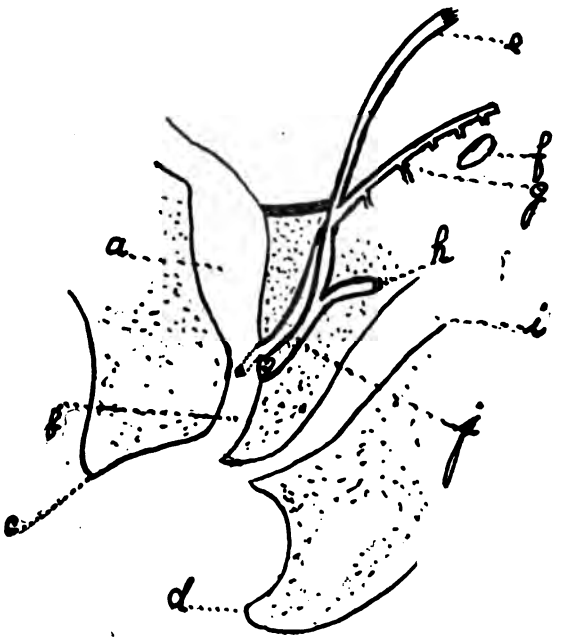

FIG. 8—Diagram of genital ducts at third month of fœtal life. Lateral view. *a*, Bladder. *b*, Urogenital sinus. *c*, Genital tubercle. *d*, Cauda. *e*, Right Müllerian duct. *f*, Genital gland. *g*, Wolffian duct. *h*, Left Müllerian duct. *i*, Rectum. *j*, Genital cord. [Modified from Keith.]

structure, resulting, in the completely developed vagina, in a stenosis of the kind we are considering. This theory, while logical and consonant with embryological facts and with other forms of inclusion, accounts as well for multiple sæpta as for single ones, since it would require but a multiple inclusion instead of a single one. And it allows of the comprehension of lateral openings on the theory of Verchère that these represent the original lumina of the primitive *Anlage* of the canals of Müller.

The fact, however, that by far the greater number of reported cases of transverse sæpta possess a single central perforation—of varying calibre—is proof of the normality of the conduct of Müller's ducts in all other respects, that is, that they met and fused in normal manner, and that the longitudinal band representing their fusion and extending from the fundus of the uterus

to the hymen, was properly and normally absorbed, hollowing out the vagina and perforating the hymen as well as the abnormal sæptum, which lay in the vagina. Nor does the fact that in a few cases the hymen appeared to be missing, and what was assumed to be the hymen was found somewhat higher in the vagina, as a transverse sæptum, vitiate the theory I have advanced; for when in a complex organ one abnormity of development appears, a second one is by no means a rare accompaniment.

The proof of my contention would be complete were the sæpta to be found histologically of epiblastic origin. While I am inclined from embryological study to accept Berry Hart's conclusions that the epithelium of the lower third of the vagina is derived from Wolffian (epiblastic) sources, my histological researches on the transverse sæpta are not yet completed. It appears to be so derived; but until such time as I have thoroughly convinced myself of their origin from the ectoderm, I prefer to hold this part of the proof in abeyance.

From the study which I have given the cases, however, I am inclined strongly to the belief that transverse sæpta in the vagina are of epiblastic origin and are due to an inclusion by the canals of Müller of cells from the Wolffian duct after the formation of the genital cord. This theory is consistent with embryological facts, with pathological findings, and with clinical observation.

Reversion.—Turning now to lower forms of vertebrate life which possess transverse sæpta in the vagina normally, we first select the chimpanzee, which is a close progenitor of the primate man. The sheep, among the *ovidæ*, the whale, the manatee, and the dugong, among the *cetaceæ*, may next be mentioned. All these animal present deep transverse folds and reduplications in the vagina. It is quite impossible to believe that embryological remnants or defects can secure permanent residence without giving evidence of function in the represented type. In the species cited in which transverse sæpta are normally present, the exact function of thse bands is not known, although it probably has to do with the facilitation of the access of the sperma fluid to the uterus, or with a prevention of the flow of the fluid from the vagina. Darwin[10] says: "It is hardly credible that a complex part, arrested at an early phase of embryonic development, should go on growing so as ultimately to perform its proper function, unless it had acquired such power during some earlier state of existence, when the present exceptional or arrested structure was normal." We find in the first two cases reported here, that the function of the normal vagina was present. It is further conceivable and probable that if the spermatic fluid were able to be injected beyond the opening of the sæptum, the sæptum would

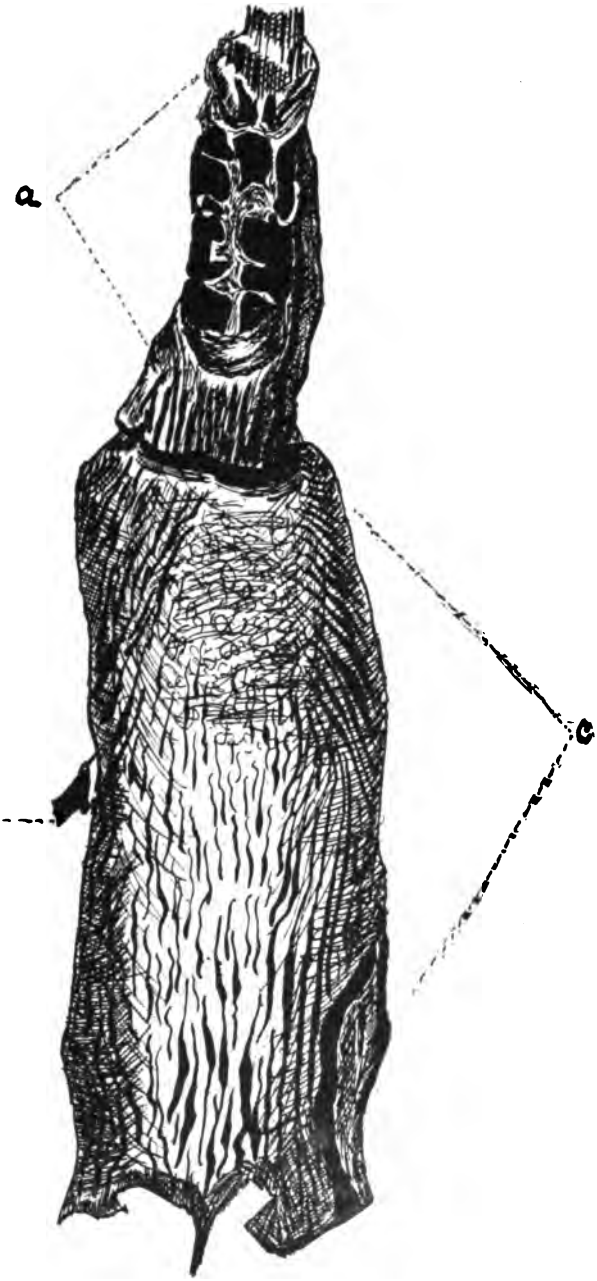

FIG. 9.—Vagina and cervix of *Ovis aries* [sheep], showing deep folds in the vagina and cervix. *a*, Cervix. *b*, Searcher in urethra. *c*, Vagina. Original specimen prepared in the anatomical laboratory of the College of Physicians and Surgeons.

act as a barrier to prevent its escape. That this actually occurs is demonstrated in the many cases of pregnancy taking place in women possessing this abnormal structure. Function—if we can speak of it in this connection—then being normal in an abnormal structure, and the structure itself being the analogon of a normal adult form in lower species, it is plain, I think, that the variation represented by a transverse sæptum of the vagina in the human species is a reversion to a former type.

Diagnosis.—The diagnosis of this malformation should offer no difficulty. It must be distinguished, however, from a *vaginismus of the superior part of the vagina* and from a *cicatricial* or *congenital vaginal atresia.* Cicatricial closures of the vagina may almost always be excluded by the history, as by the absence of previous caustic douches, injuries, and the acute infectious diseases, especially diphtheria, scarlatina, smallpox, and cholera; and, in primiparous or multiparous women, from the scarring and formation of bands so frequently seen after childbirth with inefficient or unskilful assistance. A total atresia will barely come under consideration, since the history of a regular menstruation will at once exclude it. Again, the site of the sæptum and the presence of myrtiform caruncles exclude the possibility of confounding a transverse sæptum with a persistent hymen, an error which has been made repeatedly.

During labor one must be mindful of the possibility of mistaking the sæptum for the unruptured membranes, or for a sac-like dilatation of the inferior segment of the uterus, or for an incomplete dilatation of the cervix. A careful vaginal examination, by speculum, if necessary, and catheterism of the orifice to establish the presence of the vaginal cavity above the sæptum, will settle the diagnosis in a certain manner.

The positive signs which lead to a diagnosis are the presence of the transverse sæptum discovered by the examining finger, the impossibility of feeling the cervix when the lumen is too small to admit the finger, the presence of the ringlike band which is easily felt when the finger can pass the opening, and the visual demonstration by means of the speculum.

Influence upon Menstruation and Labor.—Transverse sæpta, whether congenital or acquired, may offer obstacles to the menstrual flow and to sexual intercourse, and may thus become sources of sterility. If impregnation occurs, however, they have no effect upon the normal course of pregnancy. When labor comes on, however, they may seriously interfere with its successful completion. The factors then to be considered are the size and site of the sæptum and its degree of resistance to the labor pains, and the intensity of the uterine contractions. If it is very

tough and resistant, it may cause the death of the fœtus by the prolonged labor; and for the same reason uterine rupture may occur. Maternal exhaustion may be observed from the double work of dilating the birth passage and the obstructing diaphragm. The most frequently described accident is that of rupture of the sæptum with extensive tears into the vagina, resulting in some cases in fatal hæmorrhage. Neugebauer has collected fifty-six cases of Cæsarean section performed on account of this malformation, twenty-two of them being Porro operations. He has reported also 237 cases in which birth ended spontaneously or by the aid of forceps with the most varied result to mother and child. In most instances the child was born dead, and frequently the mother was left with vesicovaginal or rectovaginal fistulæ or with terrific lacerations of the vagina, or she died from sepsis or from hæmorrhage. Birth through the rectum in the presence of a transverse sæptum, is also recorded, the most recent one being that of Alain.[11] The prognosis, then, for mother and child, in the absence of timely treatment, is doubtful and even serious.

TREATMENT.—1. *In the non-pregnant state.* In the case of an unmarried woman who menstruates regularly through the sæptum, nothing is to be done. In the nulliparous married woman the diaphragm should be so dilated that sterility will not result; that is, if uterus and ovaries are present—for other malformations and lack of development may simultaneously appear. Coition must be rendered painless and possible to both parties. Further than this, no treatment need be instituted. If, however, the opening in the sæptum is very small and dilation cannot be practised, the sæptum may be excised and the cut vaginal surfaces carefully sutured, as done by Neugebauer[12] in 1893, and by Vineberg[13] in 1894. Dilatation with rubber bougies or with the fingers may be practised, subsequent contraction being prevented by packing with gauze.

2. *During pregnancy.*—If the condition is recognized early, excision should be done; if it is seen only late in pregnancy, dilatation should be performed, as an operation then would be too bloody as well as a possible provocative of premature labor pains. These measures have the additional advantage of permitting a thorough exploration of the vagina above the sæptum, and of allowing it to be thoroughly disinfected, for the space above the membrane has been found to be a perfect nidus of microbic residence.

If dilatation is chosen, it should be slow and gradual, and can perhaps be best achieved by Hegar's elastic bougies followed by manual dilatation.

3. *During labor.*—The plan during labor depends upon the stage of labor and its duration when the patient is seen. If labor is just beginning and dilatation can still be accomplished, it is perhaps the safest method, as infection may thus be avoided. Here the Champetier-de-Ribes's bags, or Voorhee's modification, will prove very serviceable after the opening has been sufficiently expanded by the fingers or by bougies to permit of their introduction. But if the sæptum is very tough and fibrous and does not yield to stretching, its section is the only possible measure left. Either a buttoned knife or bistoury or a pair of blunt-pointed scissors may be used for this purpose. Bilateral incisions may be sufficient to permit the birth of the child, or it may be necessary to make crucial incisions as I was compelled to do. The after-treatment consists in preventing the sæptum from forming cicatrices with the vaginal wall or with its own cut edges. Dilatation may even have to be practised after the puerperium is ended, as a prophylactic measure against a repeated stenosis.

Conclusions.

1. Transverse sæpta of the vagina are rare, occurring in about one in 5,000 cases, about .002 per cent.

2. They are derived from an inclusion by Müller's ducts of cells from the Wolffian duct or ducts after the formation of the genital cord, and are therefore epiblastic in origin.

3. Their perforation is proof of the normal conduct of Müller's ducts in all other respects.

4. Transverse sæpta of the vagina being normal in adult sheep, whales, dugongs, the manatee, and the chimpanzee, they represent in the human being a reversion, "a return to an ancestral type." Their function is purely speculative, but may have to do with the facilitation of conception; and when they appear in the human female may have a similar purpose in harmony with other minor defects of development.

5. The treatment of this condition is excision of the sæptum with suturing of the cut edges. In unmarried women no treatment is necessary. If the sæptum is first seen early in pregnancy, it may be excised; if during labor, a crucial incision will be sufficient with subsequent removal of the sæptum. The prognosis for the child is usually bad unless the sæptum is incised early or unless it is not too strong to be burst by the advancing head; for the mother, it may result in serious lacerations or fatal hæmorrhage.

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XXVI. HYDROPS TUBÆ PROFLUENS.

By HENRY D. INGRAHAM, M. D.

BUFFALO.

This condition has been described variously as hydrorrhea ovarilis intermittens, salpingitis profluens, intermitting ovarian hydrocele, hydrops tubæ profluens, as well as by other names.

Reed says in his gynecology "that this a comparatively rare condition, only isolated instances having been reported from even the largest clinics." Other writers express similar opinions. Owing to the infrequency of the disease, I believe the two cases that I have seen should be reported, even though I have not been able to verify the diagnosis by operation.

Case I. Mrs. M., age thirty-nine years; American; above the medium size, well developed and good family history. Menstruated first when thirteen years and six months old; regular, and periods normal; married at twenty-eight; one child two years later. Labor prolonged and severe; was finally delivered with forceps. Recovery tedious. She had pelvic cellulitis, and phlegmasia dolens, and it was over four months before she went out of the house, although her illness was during the summer. The child lived only a few weeks. I first saw the patient seven years ago. She had been in poor health ever since the birth of her child, now two years and a half. She was suffering at this time from endometritis, accompanied by pain and tenderness in both ovarian regions. The uterus was in normal position, but somewhat enlarged. She had a thick, tough, tenacious discharge, which was at times thinner, more profuse and often tinged with blood. From three to four days before her periods she had gushes of thin watery fluid that did not stain the napkin. This occurred from one to three times a day and as near as could be estimated she lost from six to eight drachms at each discharge making four to six ounces in all. The menstrual period which followed was normal. The same thing occurring again the next month. The patient said all these troubles followed the birth of her child; before that, she had nothing of the kind. A short time after I first saw her I dilated and curetted the uterus and mopped it out with pure carbolic acid. The thick and bloody discharge stopped, and in a few weeks she was well, except the watery discharge. This was somewhat lessened at first, but soon became as bad as before the curetting and has remained so ever since. Some months she is better but others she is worse. This was the condition before the curettage. At the time of the operation I made a thorough examination of the pelvic organ while she was under the anesthetic, and as before stated the uterus was in normal position, but somewhat enlarged. The left tube and ovary normal; the right tube appeared to be enlarged and somewhat distended. This is the only time I ever examined her under anesthesia, and it was twelve days before the expected menstrual period. I have examined her many times since.

Just before the watery discharge occurs I have usually been able to detect a small elastic mass on the right side in the ovarian region. I have not always succeeded, however, in finding the mass, when the examination was made at this time. At other times in the month I have not been able to detect anything of this character. Sometimes it seemed that the right tube was broader and folded upon itself, at least, that is the way it felt. For sometime after the curetting I applied various remedies to the uterus, used electricity and general tonics, but without any benefit. For the past four years she has had no treatment whatever, her condition remaining just about as it was soon after the curetting. Her trouble is not serious, so she declines operation, which has not been urged—merely suggested.

CASE II. Miss L., age twenty-six; American. Is rather above the average size, and well developed. For the past four years has taught school, before that was a pupil at school. Has been very constipated for eight or ten years; the past four years usually going three or four days, and occasionally a week without a movement of the bowels. Has had a great deal of headache, otherwise healthy. She began to menstruate at fourteen, regular, flow normal, and only occasionally during the past six years slight pain for the first day or so. Has never had leucorrhea. About ten days before each menstrual period for the past three years she had experienced a sense of fulness and discomfort in the abdomen, most pronounced on the left side. These symptoms gradually increased until usually the third day before menstruating. She would suddenly have a profuse water discharge, which would wet through two or three large napkins, that she wore at this time, her underclothes, and often her dress. She always knew for an instant before the discharge occurred that it was coming, but the warning was not sufficiently early to allow her to avoid wetting her clothes, even if she were where she could do so. There was no color and usually no odor to this discharge. Sometimes there was only one discharge, but oftener, there would be one or more for each of two or three days in succession. The patient estimated that she usually lost a quart or more before each period. The sense of fulness would disappear, and she would have no further trouble until the next month when the same thing occurred again. I first saw the patient early last June, a few days after her menstrual period. Examination showed a perfectly normal uterus and nothing unusual could be detected in either ovarian region. The examination was without anesthesia. I gave the patient a tonic, a laxative, some advice in regard to her diet and habits, and strongly insisted upon the necessity of a free movement of the bowels every day. I requested her to report again just before her next menstrual period. She lived twenty-five miles from the city and was unable to do so, but wrote me after the period that her bowels had been regular and for the first time in over three years she had menstruated without any watery discharge preceding it. This was a very happy surprise to the patient as well as to me. I have seen her occasionally since, but have not changed the treatment except I stopped the tonic. Before the October period she allowed her bowels to become constipated and had a return of the watery discharge, although not as much as formerly. This is the only time her bowels have been constipated,

and the only period preceded by the discharge since last June. Twice besides this she has had a feeling of fulness on the left side, but not very pronounced. I still have the case under observation, and am watching it with a great deal of interest.

I feel quite sure that the cause of the disease in the first case was due to childbirth, and the complications that followed. In the second case I think it due to the long period of persistent constipation.

Anything that I might say upon the pathology of those two cases would be mere supposition, and the time allowed this paper is too limited to enter into the consideration of the pathology of this condition in general. Yet, I might briefly state that I believe the abdominal end of the fallopian tube on the right side of Case I. and on the left side of Case II. were completely occluded by the inflammatory process and that the uterine ends were temporarily closed chiefly by hypertrophy of the muscular walls of the uterine end of the tubes and the thickening of the epithelial lining.

As a result of the inflammatory process an excessive amount of serous fluid was secreted and retained in the distended tube until the pressure became so great that the inner end of the tube opened and the fluid was drained through the uterus. Some "investigators have attributed this condition to a stricture of the tube, which, like the kinked garden hose is only overcome by the gradual increase in pressure behind the point of obstruction."

I would refer to the list of authors appended for what little further information there is upon this subject as far as I have any knowledge.

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XXVII. THE PREVENTION OF PUERPERAL ECLAMPSIA.

By JOHN T. WHEELER, M. D.
CHATHAM

A very heavy weight of obligation rests upon the physician to diagnose renal insufficiency in the pregnant woman at an early stage of the disease, while it is not only possible but comparatively easy to protect her from the serious consequence of profound toxæmia.

The disease is not an obscure one in respect either of physical evidences or clinical symptoms, if the physician is on the look out for them; to the patient and her friends, however, its early manifestations are not recognizable as danger signals, and quite too commonly the first intimation of the existence of the kidney affection is the convulsion that quickly precedes the fatal issue. So it comes about that the kidney disease of the pregnant woman has come, in practice, to be regarded from two different points of view. First, as a renal insufficiency, known only to the initiated, occurring in the dutiful patient of the wary obstetrician, recognized, in its early stages before intoxication has become portentous, and while time and opportunity offers to rescue the mother, and also the child, from impending peril.

Here the physician is free to apply the resources of his act in instituting a protracted, painstaking investigation of the woman's capacity for excretion; he makes frequent estimates of the elimination of urea, and other kidney waste, notes the presence of kidney detritus, detects the first faint warnings of autointoxication, and employs such means for increasing elimination as the necessity of the case from time to time requires. In the great majority of instances, he will carry his patient to term and through confinement without resorting to vigorous measures; or, if the disease proves intractible, he may hasten delivery under conditions of his own choosing. Being forewarned, he is forearmed. His methods are highly scientific, and, if he finds his work laborious and exacting, he has success for a reward.

I know of only one considerable series of cases reported illustrating the success attained in preventing eclampsia by observing in a routine manner the perfection of excretion. These are given by Dr. E. P. Davis of Philadelphia, and cover a period of six years. There were 1,566 patients. Of these three had eclampsia. In neither case were the convulsions severe, and in each mother and child recovered. So much for the one point of view; now for the other.

In the other class of cases there has been no observation of the functional activity of the kidneys, and the woman is left to her fate. Insufficiency once begun has gone on unchecked; the toxins accumulating in the blood, slowly at first, and declaring themselves by symptoms obvious enough to the initiated, have acquired at length an overwhelming virulence; the excretory functions are paralyzed; and suddenly, in every third case, the catastrophy proclaims itself with all its appalling significance.

There is an ironical suitability in the designation of this class of cases by the name of the symptom that comes in order last of all before death comes. We call them cases of puerperal eclampsia. It is as though we had ignored pneumonia for a week and then named it the crisis.

If the physician can look back with complaisancy upon his successfully managed cases of renal insufficiency, he will look forward with as little satisfaction to his encounter with puerpual eclampsia, as to any experience in all his troubled life. That day, whatever else his plans may have been, he has his work cut out for him. It is no time now for nice determination of future risks and careful adaptations of measures of relief. There is instant demand for violent and heroic treatment, with the certain knowledge that the best result he can hope for is, on the average, an even chance of saving the child and two chances out of three of saving the mother. Even he does not know that the doom of his patient was not sealed before the convulsion appeared. He will, if he follows the approved method of treatment, overwhelm his already semi-unconscious patient with anesthetics and narcotics, to suppress the convulsions; he will depress her still more with veratrum viride; for purposes of elimination, he will bleed and sweat and purge, and ply her unresponsive kidneys with powerful diuretics; and finally, he will hasten her delivery by rapid mechanical violence. Such treatment is justified by necessity, but it carries with it an incalculable measure of injury.

It is not my purpose to consider treatment of eclampsia, nor indeed to go deeply into the technical details of renal sufficiency, but rather to bring these two classes of cases of one and the same disease in sharp antithesis, in order to study the peculiar circumstances and conditions which determine their grouping, and to see, if, out of this inquiry, we may find a way of striking a new line of cleavage between them, so that the tragedies in the one may be fewer in number, and the triumphs of the other more numerous.

Here are two stages of the same disease, the one recognized early, the other late; the mortality of the one, in such hands as

Dr. Davis' is nil; that of the other thirty-three and one-third per cent. The treatment of the one, conservative, scientific, appealing to the humane instincts; the other, justified by dire necessity, but barbarous, unscientific, unprogressive, revolting. Except for the use of the saline solution, there has been no improvement in the treatment of eclampsia in thirty years, nor does there appear to be promise of improvement. Relief does not appear to lie in that direction.

The tendency of the disease to run on to a calamitous issue is well understood by us, the desirability of its early detection and treatment, is generally appreciated. Still the fact remains, that this acknowledged opprobrium of medicine is an almost constant quantity in our experience. I meet with it every year, and so does every other medical man in general practice.

The constructive problem seems to be, seeing that eclampsia cannot be cured, but can be prevented when recognized early, what more can be done than we are doing to make more general its early recognition? Ignorance of the nature of the disease on the part of the laity is the chief reason, among a number, why the pregnant woman goes on to her fate unprotected. There seems to be not the least bit of familiar acquaintance with the disease passing current among women, as there is in the case of most other diseases. It takes a trained observer to interpret the symptoms—hence women too poor to call a doctor before confinement, furnish the largest percentage of cases of the advanced disease. The same ignorance operates to the disadvantage of women under professional observation. Also it makes them fatalists. They do not cooperate with the doctor in his investigations, as they would if they could understandingly observe the developing stage of the disease themselves, and could have their interest enlivened by a present sense of fear. It is the policy of the doctor commonly, not to permit his patient to be disturbed by unpleasant forebodings, and for these various reasons many a well intended plan of routine observations miscarries. An intelligent, methodical patient will comply without question to the requirements of an adequate supervision, but in general, taking people as we find them, medical authority alone is not strong enough to invariably secure the necessary cooperation, because the woman does not herself sense its importance. She does not know that she is sitting on a powder keg with the betting one to sixty that it will explode. With this ignorance to blind her to danger, many trivial circumstances operate to her jeopardy. I saw a fatal case in Ghent last year where change of residence was the agency. The woman had recently moved from Troy; she did not wish to call a physician

in Troy because she knew another would have to attend her in Ghent. Her modesty probably cost her her life. Inaccessibility, as in the country with bad roads and bad weather, interfers with systematized inspection.

What now in the personal equation of the physician in the matter? It would be well if we asked ourselves with every case of eclampsia—whose fault is this? Our duty, as at present understood, is to prevent it, but we are not accountable for cases culminating outside our purview, nor when our efforts have been thwarted by the patient's negligence, although it would be greatly to our interest to lessen their number.

But can a man, whose time is fully occupied in general practice with its varied responsibilities and with the usual proportion of confinement cases, be expected to give that thorough supervision of every one of them throughout the last half of gestation that would be requisite to secure such a complete measure of prevention as Dr. Davis' series implies?

Are we never excusable for leaving a specimen of urine waiting for examination until it spoils? Considering how rapidly auto-intoxication sometimes fulminates, who can say just how frequently we should estimate the urea, instead of test for albumen?

But can we quite acquit ourselves of the imputation of too often trusting entirely to luck under the temptation of sixty chances to one against eclampsia, and two hundred to one against a death from it? Is it not true, that the disposition to take one's chances is a very common practice, and that all of us occasionally, and many of us frequently, and some of us always, enter the lying-in chamber with no information whatever of the patient's kidney function? Can we claim at all that our conscience toward uræmia is as clear as the conscience of the surgeon is toward sepsis?

It has been proved by Dr. Davis, that, from methods comparable in thoroughness to those employed by the modern surgeon, equally brilliant results may be secured by the obstetrician, but as yet Dr. Davis' experience appears to be an isolated one. Evidently the family physician and the obstetrician do not now stand upon the same high plane with the votaries of aseptic surgery. Why should they not?

Having passed now in review the circumstances and conditions which create difficulties in the way of prevention of eclampsia, I offer these observations.

I consider it a radical error on the part of the profession to permit the laity to remain in ignorance of the nature and tendency of the kidney disease of pregnancy. Since they have no

means of acquiring the acquaintance subjectively, the woman should be taught it. They should be made to understand its insidious nature, and their own insecurity, and to emphasize the instruction by an object lesson, and to provide them with at least one obvious danger signal, they should be taught to examine their own urine at stated intervals for albumen. They should have all the comfort that the absence of albumen under the circumstances would indicate, and feel all the concern which its presence might imply. They should make the test twice a week from the sixth month onward, and be ordered to notify us when the test ceases to be negative. It will not be claimed that a woman who knows when an egg is cooked cannot test for albumen.

To the objection that it will make the woman nervous, we may plead the higher need; to the objection that she will be careless, we may offer a "not proven." It would relieve us of much drudgery, which we do, or do not, perform, but it would not absolve us entirely from oversight.

If a familiarity of this kind with the test for albumen became general among woman, so that it extended to the poorer classes, it would bestow the blessing of prevention upon the one class now utterly deprived of it, and otherwise utterly inaccessible. Its ultimate diffusion and adoption, as a general practice, is as possible as would be the interest now felt in maternal impressions, if that were new.

Finally, that it is practicable, I can offer an experience of many years. I began it twenty years ago, with a woman whose six successive pregnancies had ended in convulsions at from three to six months. I found supervision particularly irksome, because she began so early, and would develop a toxic explosion within three days. With the seventh pregnancy, she began the test for herself in the second month, and continued them satisfactorily until the middle of the fifth month, when she got the warning which she well understood. After that I conducted the examinations. In this instance, if the woman had not made the daily examination herself, she would have miscarried again. Labor came on after a hot bath in the 35th week, and was normal, the child living.

I always teach my cases to make the test, if they reside at a distance, or are poor, or there are any other obstacles to personal supervision. I find the women do not neglect it, and they help to keep me up to the mark. I have known of the practice extending beyond my patients to their acquaintances, and have had women come to my office for test tubes whom I did not attend.

I am disposed to believe if the practice were encouraged by the profession, it would gain a foothold among the people, although I dare say it might develop some abuses and limitations of usefulness.

I may say I am optimistic in regard to the growing interest in the profession concerning prevention, and I think the time is not far distant when we shall hold ourselves, and be held, responsible for the occurrence of any preventible eclamptic attack.

I see no reason why, in process of time, devotion to a high ideal in this particular, under a new standard of professional obligation, should not reward our zealous efforts with a close approach to complete extinction through prevention of puerperal eclampsia. And when we shall have done all that, we shall have done our whole duty by our patients and ourselves.

XXVIII. THE TREATMENT OF PURULENT CONJUNCTIVITIS.

By EDGAR S. THOMSON, M. D.

NEW YORK

My purpose in presenting to you this brief paper is not to discuss the well-known treatment of this disease but rather to dwell upon some of the new methods which seem to be establishing themselves permanently. The first step in the treatment of every case of purulent conjunctivitis should be the microscopical examination of the pus, by slide examinations, or by cultures, if necessary, so as to ascertain the variety of the micro-organism present. Treatment need not, and should not, be delayed on this account, as in many instances every hour is of moment; but certainly no case can be intelligently treated without this information.

In cases that have the clinical appearance of gonorrhea strong germicides should be at once applied, while in cases of less severity, and presumably caused by less virulent pus germs, it would certainly be a mistake to make the same application. In fact, the selection of the germicides is a matter of no little tact. Many effective germicides cause a certain amount of traumatism to the conjunctiva, although in some recent preparations this is reduced to a minimum, and we must consider carefully whether the effect we wish to gain is worth this disadvantage.

The gonococcus is the most virulent germ we have to deal with in the eye, leaving aside the rarely occurring diphtheria bacillus, and the clinical picture is apt to be suggestive. Almost every one is familiar with the brawny, swollen lids, the

chemosis, the profuse, thick discharge of creamy pus—all coming on with great rapidity after infection, whether occurring in the adult form, or in the modified form in the newly-born infant. The disease varies somewhat, but in the great majority of cases is considerably more severe than the worst cases caused by other infections. In fact, I have come to doubt the existence of the so-called "mild cases" of gonorrheal conjunctivitis, after the examination of a large number of cases occurring in the contagious wards of the Manhattan Eye and Ear Hospital during the last five years, as well as those occurring in my own private practice.

It is reasoned by many that we should find mild infections in the eye as well as in the genital tract, but the clinical fact remains that in mild cases of conjunctivitis we do not find the gonococcus. Such cases are often called gonorrheal on insufficient evidence. Indeed, it is too often supposed that the eye condition is gonorrheal without any examination of the pus. In gonorrheal cases the treatment should be first of all directed to the destruction of the gonococcus, and our best means to this end is by the application of nitrate of silver. None of the new germicides seem capable of replacing silver entirely, and they are of use chiefly in cases where silver is not well borne. It is imperative in adult cases that the application should be made at the earliest possible period after the infection. As soon as we see a case a slide examination should be made and silver applied, as on this point often depends the success or failure of the case. If the gonococci once have a chance to penetrate the conjunctival tissue thoroughly, the eye will probably be lost, as it is impossible to reach them by germicides, and the process goes on until the cornea ulcerates. In babies, the results of delay are not apt to be so serious, as the cases are more manageable, but in every case the silver should be applied as early as possible.

When we see the case before the lids are much swollen—a period of a few hours in adult cases—it is sufficient to apply a two per cent. solution. This should be done by a swab of cotton on an applicator. The eye should be cleansed, and the applicator carried entirely through the cul de sac under the upper lid; then the lower cul de sac is to be swabbed out in the same way, so that the silver is applied to every part of the conjunctiva. This, of course, cannot be done with children. We have to be satisfied with a less thorough application, which, however, will usually be sufficient. A certain amount of reaction invariably follows, but this can easily be controlled by iced cloths, and the irritation is of small importance compared with the seriousness of allowing the gonococci to penetrate the conjunctival tissue, which

they always do with great rapidity. No other germicide is so much to be depended upon in this connection as silver. If the gonococci have penetrated, the swelling will have begun and the discharge will be profuse, and stronger solutions of silver may be necessary. Where the disease is well developed, as it is apt to be by the third or fourth day after infection, the two per cent. solution gives the best results; but before that time stronger solutions may abort the process.

It is impossible to lay down any rule as to the limits of the time when the abortive plan of treatment may be used, but my own practice is always to attempt it wherever there is any prospect of success. If I see a case two days after infection, and the swelling or discharge seems not to be at the greatest height, I apply a three per cent. or a four per cent. solution of nitrate of silver as thoroughly as possible, and then meet the further indications as they arise, using two per cent. as a daily application. It is best in using a stronger solution than two per cent. to neutralize with salt solution on account of the danger of staining the cornea, or even to lay a pledget of cotton soaked in salt solution upon the cornea while making the application. Where silver irritates, as shown by the increased discharge of serum and a tendency of the conjunctiva to bleed readily when handled, a six per cent. solution of protargol may be used. It has been claimed for this germicide that it is without irritating properties, but this is certainly not so. It is, however, very much less irritating than silver, and many patients do not feel the application of a six per cent. solution at all. Stronger solutions are of no particular advantage,—the irritation is more marked and the germicidal properties seem to be not appreciably greater. Protargol is a valuable drug in ophthalmia neonatorum in cases where the conjunctival thickening is not great enough to support applications of silver. It may be dropped into the eyes twice daily.

In other forms of purulent discharge, that is, those caused by the pneumococcus, Koch-Weeks bacillus, or other pus organisms, silver is not to be used, but protargol in six per cent. solution applied once a day is a most efficient germicide, and will often limit an attack of "pink eye" to a few days, which otherwise would last two or three weeks. Protargol has the further advantage of not staining the tissues as readily as silver; and it is good practice to flood a corneal ulcer occurring in the course of a gonorrheal conjunctivitis with a six per cent. solution once a day. It has been claimed that protargol will not stain the conjunctiva but cases have been reported in which its prolonged use has led to a slight stain. The claim at first made for it that

it will penetrate the tissues and destroy germs more deeply than silver, seems not to be borne out by clinical experience. In severe cases of gonorrheal conjunctivitis with papillary hypertrophy the astringent action of the silver is to be desired, and the discharge subsides less readily under protargol than under silver. Argyrol, 25 per cent., or silver vitelline, fulfills all the above indications for protargol, and is absolutely non-irritating. It also is less effective than silver where an astringent is required; but where a non-irritating germicide is required, it is certainly ideal. I have recently seen it injected into the anterior chamber of an infected eye with remarkable checking of the process and without any irritation to the iris whatever.

The other indications for treatment in purulent conjunctivitis are best met on the old-fashioned lines. Constant cleansing with saturated boracic acid solution resists the development of corneal ulcers and keeps down the number of germs. Boracic acid is merely a non-irritating wash and has no germicidal value. If we use plain water on the conjunctiva or any other mucous membrane, there is a tendency to exosmosis set up at once in the blood vessels, which dilate—a phenomenon frequently observed when the eyes become red after being filled with tears in weeping. The least irritating solution, therefore, is one which has nearly the same specific gravity as the blood. The cleansing should be done often enough to keep the eye clean, even if that is every ten minutes day and night.

For the swelling of the lids, iced cloths applied constantly are best, and these have another very important action, as has already been pointed out, in reducing the temperature of the conjunctival cul de sac below the point at which pus organisms grow best. It is possible to reduce the temperature several degrees, while heat cannot be applied constantly, and most pus germs bear slight degrees of increased temperature better than diminished temperature.

Corneal complications are to be met in the usual lines, the only advance in recent times being the use of protargol or argyrol, either of which will destroy the germs in corneal ulcers as well as in the conjunctiva, and that without the irritation and danger of staining which makes the use of silver in this connection impossible. In a certain small proportion of cases, where the chemosis is severe and the discharge not free, scarification of the chemosis is of value, notwithstanding the fact that it has fallen into disrepute of late years, the reason for this latter fact being that its advocates at first claimed a much wider field for its application than was justifiable, as is so commonly the case with methods and remedies in this disease. Scarification will

not reduce every case of chemosis, by any means; but in certain cases where the discharge is scanty and the chemosis is of a boardy character, scarification will relax the chemosis in a way that no other means will accomplish.

Cauterization of the cornea, in cases where the chemosis is not severe, is a most valuable means of combating corneal ulceration, and is particularly useful in ophthalmia neonatorum. Pure carbolic acid is the best cauterizing agent to use. It need hardly be said that we should be reasonably certain that the tissues will react well to the cautery, which will not be the case if the chemosis is very severe, or if the patient's general condition is much reduced. When corneal infection appears, hot water applications are to be used for the purpose of stimulating the corneal tissues.

XXIX. TRANSPORTATION AND THE OPHTHALMIC REFEREE.

By JUSTIN L. BARNES, B. S., M. D.
NEW YORK

On both land and sea the importance to the public and to transportation companies as well, of thorough testing of the sight, color-sense, and hearing of operating employees, can scarcely be overestimated. In Sweden, in 1875, an engineer passed a danger signal and hurled his train upon another, resulting in loss of life and property. The affair was shrouded in mystery, until Holmgren, suspecting the accident might have been due to color blindness, pursued investigations and confirmed his suspicions, which finally led to the general adoption of color-sense examination, of more or less perfect character, in nearly every civilized country in the world.

In the United States, within a couple of years of this writing, and notwithstanding the fact that color testing is in this country widely practised, similar accidents have occurred. Naturally, the public is ignorant of this fact. That after a lapse of about a generation, such an occurrence can be ascribed to deficient color sense, seems impossible and the questions naturally arise: How is this to be explained? What is the solution of the problem? How is the public safety to be guaranteed? The plain answer is that even with the best intent the subject is generally but superficially understood, and the importance of really rigid and repeated expert investigation of the color sense of employees is greatly underrated. This is certainly shown by the fact that among American railway companies we have all grades of effic-

iency in color testing, from the well-nigh perfect systems of certain roads to the line that is "going in for something of the sort shortly."

The Committee of the American Ophthalmological Society on Standards and Methods of Examining the Acuteness of Vision, Color Sense and Hearing for Railway and Marine Service recommended, among other things: "That a trained ophthalmic surgeon be selected by each company, who shall instruct and examine the man selected by the company to make these tests, shall recommend the standards and methods to be used, shall see that the equipment furnished to each examiner is sufficient, that it is kept in proper order and renewed when necessary, and who shall be the *authority to whom doubtful cases shall be referred for final adjustment*" (italics mine). This recommendation was adopted by the society.

In order to obtain as clear a view of the situation as possible, I have been in active correspondence with fifty leading railway companies of the United States and Canada, submitting to them, under the promise of professional confidence, the following letter and accompanying form of questions.

DEAR SIR:—I herewith request your cooperation in preparing an article for a medical journal on the subject of eyesight and hearing among railroad employees. If you should feel disinclined, let me urge upon you that, if so indicated, your reply will be strictly confidential, the code of medical ethics guaranteeing entire respect for your expressed and implied wishes.

I will thank you if you will kindly and promptly fill out the enclosed form, and mail it to me with as little delay as possible. If this subject is not within your province, will you please refer my letter to the proper officials? Yours truly, etc.

EYESIGHT AND HEARING ON AMERICAN RAILROADS, DEPARTMENT OF EXAMINATION

1. Do you employ an oculist and aurist? 2. If so, whom (name and address)? 3. If not, whom (name, address, capacity in which examiner is employed)? 4. What methods are used in testing—(*a*) for sight? (*b*) For color perception? 5. Do you record findings (please send specimen blank form)? 6. Is the ophthalmoscope used? 7. Is each eye examined separately? 8. Is any test made to prove that an employee sees simultaneously with both eyes? 9. Are all employees as well as candidates examined? How often (if more than once)? 10. Have you discovered any who have color blindness? 11. About what proportion have you rejected for—(*a*) defective vision? (*b*) defective color-sense? 12. What methods are used for testing hearing? 13. Any further information?

The recommendation, above noted, is certainly justified and the importance of a referee, both to the public and to the companies concerned, is plainly shown by an analysis of the statistical table prepared from the replies received.

Tabulation of Replies to Dr. Barnes's Circular Letter.

Table.	Examiner.	Sight tests.	Color tests.	Hearing tests.	Records Kept.	Ophthalmoscope.	Exam. of each eye separately.	Binoc. vison tested.	Frequency.	Rejections for C. B.	Rejections for defective vision.	R. Rs. addressed.	Replies.	Percentages.	Remarks.
Lay	7	..	..	..	..	..	..	..	..	..	..	..	..	22	Variable efficiency.
Medical	21	..		..	..	..	..	..	..	.			..	66	Rely chiefly on laymen.
None	4	4	4	4	..	..	..	..	..	..	..	..	..	12	Some chiefly contemplating.
Snellen, Jaeger, etc	..	28	..	..	..	..	..	..	..	..	..	..	..	87	Probably well done.
Holmgren	..	..	26	..	..	..	..	..	..	..	..	..	..	85	Probably well done.
Scripture's Dial	..	..	1	..	..	..	..	..	..	..	.	..	..	3	Not in common use.
Lanterns and flags only	..	..	1	..	..	..	.	..	..	..	..	..	..	3	Very unsatisfactory.
Watch and voice	..	..	..	26	..	..	..	..	..	..	..	..	..	85	Variable efficiency.
Acoumeter	..	..	..	2	..	..	..	..	..	..	..	..	..	6	Note small number.
Filed	..	..	..	..	26	..	..	..	..	..	..	..	..	85	
Not filed	..	..	..	..	6	..	..	..	.	..	..	..	..	20	"Except when defective."
Used	..	..	..	..	..	13	..	..	..	..	..	..	..	40	
Not used	..	.	..	..	..	19	..	..	..	..	..	..	..	60	Note large number.
Yes	..	..	..	..	..	..	23	..	..	..	..	..	..	70	This means no Snellen types.
No	..	..	..	..	..	..	9	..	..	..	..	..	..	28	Probably means no test of B. V.
Yes	..	..	..	..	..	..	..	10	..	..	..	..	..	31	Uncertain efficiency.
No	..	..	..	..	..	..	..	22	..	..	..	..	..	7	Note large number.
Once	..	..	..	..	..	..	..	..	10	..	..	..	..	31	About once in 3 years.
More than once	..	..	..	..	..	..	..	..	18	..	..	..	..	38	About once in 3 years.
Percentage	..	..	..	..	..	..	..	..	..	3%	5%	..	..	..	Replies very doubtful.
	..	..	..	..	..	..	..	..	..	..	..	50	32	..	

The essential question is: "1. Do you employ an oculist and aurist?" This was done to learn whether the several companies relied upon lay or medical advice. Of the fifty companies addressed, full replies were received from thirty-two; and of these, twenty-one state that they retain a medical expert (ophthalmologist) for ensuring accurate examination of sight, color sense, and hearing, while the remaining one-third confess that they do not make use of such advice, but either resort to examinations by officials of their own lay bodies or are "contemplating the idea of examinations." It is probably safe to assume that corporations not replying do not resort to a medical referee. A curious feature of this subject is the fact that most of the companies which do not retain a consulting medical expert referee are among the older and eastern companies (with one very notable exception), while those that do rely upon such referees are commonly western, newer, and progressive companies.

Probably, I can in no better way demonstrate the importance of the committee's recommendation than by commenting upon the usual methods now in force, especially among those transportation lines which rely upon lay examinations and judgment.

(I) *The Examiner.*—The examiner should, properly speaking, be a medical expert, but as probably no one oculist could be found with time enough to examine all the employees, even of a single line, a trained ophthalmologist should nevertheless be retained as a referee. However, as at least one-third of American railroads rely wholly upon lay advice, and inasmuch as this paper, not being a treatise on sight and hearing, will deal neither with the science of color perception, nor with a history of the study of color sense or color blindness, I shall confine my efforts to a demonstration of the necessity of accurate and scientific testing.

The lay examiner, upon whom the public safety depends to no little extent, is commonly a yard master, who, be it said in justice, though usually first subjected to an examination himself, may never have been competently examined, if at all. On one prominent eastern railroad the last examination of the men was conducted with wools, test cards, flags, yard lights, and the voice, certainly a primitive outfit, and by a conductor, who, in the nature of things, should not, in legal phrase, have been "competent" to referee a doubtful case, particularly in the case of old employees. This examiner, being a layman, cannot use the ophthalmoscope or perform more than the very perfunctory tests ordered by "the company rules and regulations." He could not distinguish between congenital and acquired color blindness, or diagnosticate the often temporary and curable loss

of central vision due to certain toxic influences, such as alcohol or tobacco, the result being that a dangerous man might be overlooked or a temporarily disabled but good man might be returned "not satisfactory;" while, on the other hand, it might require argument and proof to convince a superintendent that a man whom he had examined and declared "satisfactory," might, after all, be color blind. It is difficult for him to believe he has not learned or cannot be taught colors; yet a scientific examination will prove the matter.

(II) *The Conditions.*—While the examinations should be conducted with every endeavor to safeguard fairness to the employees, it must be remembered that they should be held, not alone in the interests of the men or of the companies employing them, but of the public at large, from whom transportation lines derive their support. Therefore, the examiner should see that the condition of light, materials, instruments, test cards, lanterns, flags, etc., is *uniform;* that there shall be no hurry in the examination; that he takes into consideration the nervousness natural to an ordeal which is fraught with vital significance to the man before him; that a careful *written* record is made and *filed* for reference, to which appeal may be made; that all possibility of deception or unfairness shall be eliminated; that no unnecessary persons be allowed in the examining room; that sufficient, intelligent variety in the tests be prepared to prevent a dishonest man from competing with an honest one; and that, above all, every doubtful case shall be reported with a recommendation that the examinee be sent to an expert ophthalmologist.

(III) *The Outfit.*—This should consist, as a minimum, of several cards of Snellen's test types, or an assortment of cards bearing single lines from the same; cards bearing, in sizes corresponding to the Snellen types, the various signals shown by the "semaphores;" Jaeger test types; a case of trial lenses, including spheres and cylinders, prisms, colored glasses, etc.; perimeter; phorometer; ophthalmoscope; the ophthalmometer; the Hirschberg double spectroscope; the Stilling charts; the Holmgren wools; the cobalt blue glass; the Williams's lantern; the flags; the Kroll charts and stereoscope; the acoumeter; the stopwatch; the aural mirror; specula, etc.

The whole of this outfit must be kept in the best of order, guarded against exposure to light when not in use, and replenished from time to time. It should be accessible only to examiners or officials.

How much of this outfit can the lay examiner use? It will be

found that he limits himself at best to the Snellen and Jaeger types; the + two-dioptre-lens spectacles and plane glass spectacles; the semaphore cards; the Holmgren wools; the lantern; the flags; the watch (or voice), leaving an important and valuable number of exceedingly efficient instruments and tests alone.

At this point it may be well to repeat that, as the object of this paper is to demonstrate the propriety of referees in connection with transportation companies, and not to be a treatise on ophthalmology, the details of the various tests will not be enlarged upon, but the reader is referred to the well-known text books and articles, especially to those mentioned in the bibliography.

(IV). *Visual (form) tests.* (1) *Distant vision.* (*a*) This should be ascertained by examining one eye at a time, carefully maintaining exclusion of the other, by setting before the candidate a series of Snellen's test types, and also by displaying semaphore charts. These tests are commonly well done by the lay examiner; but he must vigilantly discriminate in details, such as not permitting the examinee to turn his head to one side, having a large supply of charts, or better still a series of cards, each bearing but a single line of letters, and varying the size of letters from one card to another.

(*b*) Each candidate should be examined at distance, not alone with the naked eye, but with plane glass spectacles and + two-dioptre-lens spectacles, in order to see if he can read Snellen types as well with a + lens as without. This is done to find out whether he has *hypermetropia* (far-sight) or not. If he can do this, there will come a day when he will require glasses, even for distant vision. This test is now made by many lay examiners. It is, however, no less than fair to the candidate to warn him that there will come a day when he must be taken from his engine if in youth he can see as well (or better) with a plus lens as without; and because, in later life, his failing sight can be restored only by glasses, no engineer is safe to trust with a train if he must wear glasses, owing to the liability of glasses to accident, clouding from steam, rain, snow, dust, etc.

(*c*) A test should be made to find out if the candidate has ***binocular single vision, i. e.,*** whether he uses his two eyes simultaneously. This test is not usually, in fact is rarely, resorted to by lay examiners, as is shown by looking over the replies to the query blank sent out as referred to above. This test is a most important one, and its omission is a matter of wonder and grave concern. If an engineer does not possess simultaneous vision of his eyes, it is not difficult to see to what source we must ascribe

a certain percentage of accidents. For instance, he may pass an examination of each eye, one proving better than the other. On a "run" he may get a cinder in his better eye, and, relying upon the other, which may have deteriorated from practical disuse, fail to interpret properly his signals, and in an emergency when his fireman cannot be summoned quickly enough. This test is one which should be made with great care, patience, and perseverance, and involves an experience and degree of skill not within the province of a layman.

(*d*) When the examiner finds a man who has defective vision, a careful examination of this man should, in fairness to the candidate, be made with the ophthalmometer, the trial case, the ophthalmoscope, etc., in order to learn just what lies at the bottom of his failure to come up to the standard, whether the deficiency is permanent or temporary. None of this work can properly be done by a layman.

(2) *Near Vision.*—The customary, and usually satisfactory, test is the use of the Jaeger test types, which are employed to ascertain whether a man can read train orders, bulletin notices, despatches, etc.

There would be little to criticize here in the lay examination, provided in the latter the questions of simultaneous or binocular single vision were investigated; for, as indicated above, want of binocular vision might lead to complications, though probably not to such a degree of consequence as in distant vision.

Here the stereoscope with Kroll charts and reading through an obstruction, such as a pencil, should especially be employed, and also the phorometer.

(V) *Visual (color) tests.* (*a*) Color blindness may be temporary or permanent, acquired or congenital. Of congenital color blindness, I may say at once that no way of acquiring color perception is known, which is true also of certain forms of acquired color blindness. The acquired forms may be temporary, as in hysteria and the amblyopia (defective sight) of users of alcohol or tobacco, or may become permanent. A man who has been proved to have perfect color sense may lose this faculty, especially if he is a consumer of tobacco or alcohol; and the very essential danger of a lay examination is that such a man may pass successfully, in the examining room, matching colors correctly, interpreting lantern signals perfectly, and doing all this through correct interpretation of his eccentric vision which he can employ in proportion to propinquity; when, on the road he is liable to confuse distant color lights, because, at and in proportion to distance, the images of the lights fall upon the very por-

tion of the retina, governing central vision, affected by the toxæmia.

A very prominent cause of a gradual and insidious process by which the optic nerve degenerates, leading to defective vision and color sense, is syphilis—beyond a certain stage of which there is no hope of saving a man his position of responsibility.

(*b*) The essentials of color sense testing lie, not in naming colors, but in matching them, and in being sure that a candidate really matches through a sense of color rather than by keen observation of differences in illumination, and whether by yarns, lights, flags, or spectra.

For these tests the Holmgren wools are very properly and commonly used by lay as well as medical examiners. Now it is a curious and interesting fact that a man color blind may acquire a truly remarkable accuracy in distinguishing colors, by noting their differences in degrees of illumination, and may point out under favorable conditions in the yard the red, green, and white lights of the various signals. This is done through careful comparison, but is the more unreliable because changes in atmospheric conditions, such as fog or smoke, may be sufficient to make a man think he sees a light of a different color from the actual one, or if of slight degree of obstruction may cause him to name the color correctly when really color blind. Moreover, such interposing obstructive media may subdue the intensity of light from a color signal to such a degree as to be incapable of perception by a man of feeble color sense. In the yarn matching test, such a man is found to select in addition to correct colors, those of the confusion class, thus proving himself color blind.

The difference in degree of efficiency between the two classes of examiners lies in the personal equation, very much in favor of the ophthalmologist. For instance, ask any official if he would be willing to trust his life to an engineer who can tell signals only by their difference in intensities of illumination; or if he would be willing to sit behind an engine driven by a man before whom, for argument, we will say, there have been placed for signals uncolored lanterns varying only in intensities of light? A prompt negative will follow; and yet this engineer may have successfully passed the lay examiner. Furthermore, I can assert that any examiner who relies upon the very valuable Holmgren test, a test which should never be omitted, a test upon which the lay examiner chiefly relies, takes upon himself a responsibility, albeit ever so unwittingly, to which may be serious consequences. For, as indicated above, the man who can match yarns with his eccentric vision, may have an uncertain central vision, and yet

may have fairly passed his distant test for form vision—which test might naturally be deemed sure to exclude possibility of overlooking central scotoma (blind area). Again, the fact that a man can tell yard lights proves nothing; it is merely confirmatory. If he is color blind and carefully distinguishes degrees of luminosity, he can acquire an accuracy in interpreting signals which might be reasonably reliable, if the atmospheric conditions were constant; but as they are not, in varying atmospheric conditions a man who is color blind is sure to fall into some disastrous error.

The lay examiner flatters himself he gets around this difficulty by interposing media of greater or lesser obstructive character; for instance, in testing with colored lanterns. This is proper and satisfactory so far as it goes; but, it does not eliminate the dangerous element of a central scotoma, the eccentric vision of which is capable of detecting colors in the short range of an ordinary yarn examination. The remedies for this defect in the examination are numerous, all of which have as their object the compelling of an exhibition of what the examinee can do in the way of *central* vision. So far as I know no railroad has attempted, through lay examiners, to accomplish this object, nor has any company apparently known of its importance or understood its full significance.

Another important matter is the fact that only one eye should be tested at a time, for very important and obvious reasons. Very few companies test each eye singly, and on this account a central scotoma can easily be overlooked, and should anything happen to exclude even momentarily the better eye, one can easily comprehend the danger of trusting to a man suddenly compelled to rely upon his deficient eye. Moreover, unilateral color blindness is not unknown, and a similar contretemps would operate in the same way. The diminution and limitation of central vision are most insidious, to be feared and carefully apprehended.

(*c*) The percentage of color blind males has been found, by various and widely separated authorities, to be from four to five per cent. This means that of every twenty-five men one may be found who is deficient in color sense. The significance of this may be shown by taking the case, for instance, of one of the most prominent American ralroads. This road, with its leased lines has, according to the "Railroad Officials, No. 29," more than 1,200 locomotives, with presumably about 5,000 engineers and firemen, not counting yard masters, switchmen, etc. If no examinations were conducted at all, this would mean that there would be not less than between 200 to 250 color blind employees

to whom life and property would be entrusted. Upon this road, where lay examinations alone are held, what official can safely assert that the public may not ride behind an engineer color blind at least in one eye? If in spite of the vigilance of the lay examiner even one such engineer is on duty, the chances of accident are not reduced to that minimum which would naturally result from examinations before a medical referee, and the transportation line has not done its full duty or considered its interests until the company medical referee is an established fact.

(*d*) The test of actual signaling with service lanterns, semaphores, and flags is proper and should not be omitted, but I shall have little to say of these matters at this point, for I do not consider them of great importance, but rather as confirmatory than otherwise. The color ignorant by the way should be excluded from service where signals must be interpreted, unless, as may be, in instances where such a person proves satisfactory on testing after a period of instruction.

(VI) *Hearing tests.* The tests upon which reliance alone should be placed are the use of the acoumeter and stop watch. The voice is not a uniform agent for performing tests and such tests can not be satisfactory to anyone concerned, nor are they susceptible of accurate record.

It is distinctly important that the tests should be uniform in order to be fair to the men, trustworthy for record, and a real safeguard to the public. For this purpose the acoumeter alone constitutes the essential feature of auditory testings. The voice is a confirmatory element, but it is positively unfair to the men, as it enables the examiner to exhibit differences in uniformity which might be ascribed to partiality or disfavor, be they in fact performed never so conscientiously.

(VII) *Test Records.* It is of very great value and importance that a complete and accurate record of all tests of vision, color sense and hearing be made of each candidate and regularly filed for reference, consideration, and comparison.

For instance, suppose an accident occurs by an engineer passing a red light. It would hardly satisfy a suing plaintiff, or be to the credit of the road, for the latter to be unable to show that this man had satisfactorily passed an examination. The placing of responsibility is ever a very important matter, and a carefully prepared record would go a great way toward unraveling unknown or doubtful features of a disaster. Again, a man might be charged with defects, when an exhibition of his record might save him from a very serious dilemma, and thus result in taking the responsibility from innocent shoulders. In securing

the efficiency and honest administration of a railroad or marine service, it is necessary to be fair to the men and to place responsibility exactly where it belongs, for in this way alone can real cooperation of the various departments of the service and public satisfaction be maintained.

VIII. *Frequency of Tests.* All tests of vision, color sense, and hearing should be repeated at stated intervals on every man who has to do with motive power or signaling, and I think that the usual period for re-examination customary with some roads of "once in three years" is too long an interval. I am of the opinion that nothing is gained by such delay, on the contrary that it is a serious defect in the system. These examinations should be repeated annually; after every illness; and perhaps oftener in the case of a man who uses alcohol or tobacco. So great are the changes in the vision and hearing of occasional cases that the public should be protected against the fortuitous opportunities for disasters due to the defects in question.

IX. *The Medical Referee.* The character of the referee's examination is twofold. First, he scrutinizes the chart submitted to him to see that a proper lay examination has been made, so as to gather from it certain data for investigation. Secondly, he performs tests scientific and beyond what is within the province of the layman.

Among these tests are the use of the ophthalmoscope, phorometer, perimeter, ophthalmometer, spectroscope, cobalt blue glass, Stilling's charts, the Williams's lantern (with special contrivance to preclude eccentric vision), stereoscope, aural mirror and specula. If I have been sufficiently lucid in the foregoing pages, it should be apparent to the reader that transportation lines on land and sea should at least fortify their lay examiners with medical and responsible referees.

To illustrate further: I have referred above to the question of interpreting colored lights by differentiating their intensities without recognizing any other differences. It is a remarkable fact that many color blind can indeed pick out a red from a green light, solely by differences in intensity. Yet this can not be a reliable means of distinguishing the lanterns on the road, on account of the varying atmospheric conditions, etc., as explained in a preceding section.

But, as I have said, probably no oculist or aurist could be found, certainly not one of high and responsible standing, who could give the time necessary to examine all the men of even one line. The lay examiner should separate from the simpler those complex cases which should be sent to the referee, to whom

all doubtful cases should be referred. The referee should control the materials for the examinations and exercise general supervision over the whole function. In order to secure the confidence of the men, the officials employing them, and the public, a scientific expert would be invaluable.

(X) *The Law.* Not only is a referee a legal protection, both to employees and corporations and therefore to their interests, but also testing of vision, color sense, and hearing *should be required by State law and not left alone to private corporate decision;* and here it may be said that only three States of these United States (Ohio, Massachusetts and Alabama) do so require it. The difficulties with private corporations are very great. Here the interests and safety of the public contend with ignorance, prejudice, expense, and the incredulity of supposed immunity. Legislation is here required; for, notwithstanding the fact that about two-thirds of the number of transportation companies rely upon the ophthalmic referee, there remains one-third, a large proportion, of corporations which, failing to recognize their private interests in this respect, do not protect themselves and the public in the manner indicated.

The time should not be far distant when no railroad or marine line can be found which does not possess an expert ophthalmic referee.

DISCUSSION

Dr. PERCY FRIDENBERG, of New York: The question of color perception, which has been so admirably presented in this paper, has become of very great interest in recent years, and numerous articles have been published, not only by medical men, but by psychologists and experts in physics. One important question that arises is as to whether color blindness is preventable. Eldridge Green, an English expert, has called attention to the fact, that the percentage of color blindness is very much larger in males than in females, and also that a very large number of color blind are found among the poor and uneducated people. To-day, when color tests are made in schools, it is found that scarcely one per cent. is color blind; hence, it seems possible that with proper training of the color perception this percentage may be materially reduced in the future.

There is a growing prejudice against the Holmgren tests. The railroad men object to this examination, because they say that their work does not require them to see eight or a dozen or fifteen variously colored lights at once. They do not, however have to pick out delicate shades of color; hence, the problem presented to the practical railroad man is not the matching of colors, but merely the naming of the color which presents itself. Furthermore, it has been shown that men who can pass the wool test very frequently fail with the lantern test if the latter is properly applied, *i. e.*, if a bright red light is obscured more or less, or a light is made bright at one moment and dim at another, as it might be in practice by the turning

off of a switch. The remarkable ability to discern between differences in illumination has been commented upon in the paper. This added sense often takes the place of the one lost to such an extent that they can pick out the color wools in a way which puzzles the examiner. The men have been known to tell the color by the way the wool was dyed, and by the feeling of the wool, whereas these same men failed with the lantern tests. I think all will agree that an expert ophthalmologist should be employed as a referee, but on very long lines this becomes difficult of accomplishment. Under such conditions the examinations should be carried out by others, and the expert used as a referee. By giving the numbers of the colors on a numbered stick, and recording these on a proper blank, the charge of unfairness in the examination may be eliminated. Persons with defective color sense at the macula are able by moving the eye to pick out the colored wools, whereas with a single point of illumination the result is very different. I have recently devised a model for carrying out this kind of examination, and submit it herewith.

XXX. RETINOSCOPY.

By D. H. WIESNER, M. D.

NEW YORK

For the past decade or more, particular attention has been directed to the refractive conditions and errors of the eyes; marked relief has been afforded by the wearing of properly fitted and adjusted glasses; a long train of symptoms, classed under the general term of asthenopia—eye strain—have been alleviated, in so many instances entirely overcome; this asthenopia includes headaches, pain in eyes and temples, blurring of vision, blinking and pinching of eyelids; among school children the inability to see the blackboard work distinctly at the usual distances.

Without entering into the technicalities nor intricacies of the subject nor yet the bibliography, the purpose of the paper is to bring to the notice of this meeting of the State Medical Society some facts and plain rules and methods of retinoscopy.

Many sufferers from asthenopia apply for relief at first to the family physician; eliminating other causes, if the doctor feels the lack of proper focusing power of the eyes to be the reason of the asthenopia, would it not be to the advantage of the family physician to have a simple and yet fairly certain test to be a guide in the advising of the patient; to many general practitioners the eye is a sealed book, outside of simple anatomy. It may be that by knowledge of the retinoscopic test, one not

particularly difficult of acquiring, a new field will be opened that shall be of the greatest help and advantage to both the family physician and his patients. In conversation with a brother oculist on this matter he remarked, "that the general practioner was not educated up to the point where he could use the retinoscope intelligently to mutual advantage of the doctor and patient;" is not this a direct stigma, when it is a fact that so many so-styled "refracting opticians," "optical specialists," etc., do use retinoscopy and get results and draw deductions from them and give glasses.

The physician has knowledge of the anatomy and physiology of the eye; with patience and perseverance he may acquire enough skill and proficiency in the use of the retinoscopic mirror to enable him to diagnose the refractive condition present in the patient and direct him where he can be skillfully and scientifically treated.

The "refracting optician" has no knowledge of the physiological functions of the eye; is not fitted by study nor training to fit nor prescribe glasses, and should not be allowed to so do; this matter might be gone into more at length; but let this suffice.

The retinoscope is a piece of *plane* mirrored glass, circular in shape with an opening in the centre, and should not be more than one and one-half inches in diameter; this is mounted on a handle, preferably a long one as this facilitates rotation; the light for illumination preferably that from an argand gas burner should be behind the patient; when examining the right eye the examiner stands in front of the patient and uses his right eye—contrariwise with the left. The distance from the examined eye should be about one meter—40 inches. It must be mentioned right here that the retinoscope reveals no pathological changes or conditions within the eye; using the retina as a point of illumination the mirror only shows by its rotation the refractive condition of the eye, whether this be normal or in error; this it does by showing up the illuminated retina through the pupil, which illumination in the usual cases is round or circular; in some of oval shape; again in others showing a band of light, vertical in direction; this may also be off from the vertical to the one side or the other. This illumination we purpose to study without entering to any great length to discussing the optical reasons for the illumination nor yet the why and wherefore of the results shown by the mirror. We will take facts and findings as they are and draw deductions therefrom. Both meridians, the horizontal and vertical are to be studied.

Hyperopia. The hyperopic eye is the one shorter in its antero-

posterior diameter than that described as the normal—emmetropic—incidentally it might be mentioned that emmetropia being the normal, all conditions not so are classed under the general head of ametropia—also that the emmetropic eye is extremely rare, seldom met with, most people being born with eyes slightly hyperopic, nature evidently intending that the ciliary muscle, the muscle within the eye that by its action on the lens focuses rays of light properly on the retina, should not be inert; so seldom is the emmetropic eye seen that the slightly hyperopic eye has been considered as the normal by many. To have a standard of comparison it is better, however, to have a positive starting point, hence the emmetropic eye—the eye in which rays of light passing through the dioptric media focus on the retina without the use of any accommodation.

The hyperopic eye is the one in which entering rays of light focus behind the retina; also in which the emergent rays, and these are the rays to be studied, are divergent, which if continued to infinity would never meet. So using the mirror the illuminated pupil is circular, rotating the mirror the illumination moves in the same direction with the rotation in both the vertical and horizontal meridians; these movements are rapid, their rapidity depending on the amount of the hyperopia; very low degrees causing a very rapid movement; the higher degrees of this error producing a slower movement. The beginner may have difficulty in illuminating the retina, and in handling and rotating the mirror, and also in appreciating the showings of the mirror, but after repeated efforts one may become very skilled in the use of the test and its findings.

So the illumination moving with the rotation of the mirror in the horizontal and also in the vertical the case is one of hypermetropia.

Myopia is the reverse of hyperopia; the eye is longer than the normal; rays focus in front of the retina. Compared with hyperopia, myopia is rare in children in this country, in many instances being an acquired condition. In the myopic eye rays of light emerge in a convergent manner, focusing at some point in front of the eye which point measures the amount of myopia; now using the mirror it will be found that the movement of the illumination will be in a direction opposite to the rotation of the mirror; therefore when the illumination moves in a direction opposite to the tilting of the mirror in both meridians the case is one of myopia.

There remains another condition, astigmatism. This is a refractive error due to a faulty curvature of the cornea: so that when the term astigmatism is used it will be remembered to

apply to the cornea. To be sure, astigmatism of the lens exists, but is rare, and only mentioned in this paper.

Astigmatism may exist alone, being either hyperopic or myopic, according to a lessened or increased curvature of the cornea; it may be hyperopic in one meridian and myopic in the other, causing what is termed mixed astigmatism, and may be associated with hyperopia or myopia, producing compound hyperopic or compound myopic astigmatism. Astigmatism is in one or the other meridians, hence hyperopic astigmatism is with the rule when at 90° and myopic when at 180°—the reverse of this is met with when the astigmatism is against the rule; again, the axis may be anywhere between the vertical and the horizontal, at an off axis.

Simple hyperopic astigmatism may be recognized as follows: if of not too high a degree the illumination will be clear and distinct but instead of being round or circular assumes the shape of a band of light. Now in examining the two meridians if it be found the illumination move with the mirror in one, while in the other there seem to be or there is no movement, the condition is one of simple hyperopic astigmatism.

Myopic astigmatism may be recognized by, in many cases, the band of light rather than the round circular illumination; if now the mirror be tilted, in one meridian the movement of the illumination be against while in the other there is or seems to be no movement, the case is one of myopic astigmatism. Mixed astigmatism may be recognized by the fact that the movement in one meridian is with the mirror, while in the other it is against; that is there is a condition where both hyperopic and myopic astigmatism exists but in the opposite meridians; simple astigmatism that is unassociated with hyperopia and myopia is common, but by far the greater number of cases of astigmatism are in combination with hyperopia or myopia and then constitute compound hyperopic or myopic astigmatism.

There remains one other condition to be mentioned, antimetropia—that is where one eye is hyperopic or astigmatic with hyperopia, and the other myopic or astigmatic with myopia. Here the mirror will detect the condition when if the movement be with, the condition will be hyperopia and if against, myopia, testing each meridian separately and individually.

Just a word as to the determining the amount of error of refraction, Ametropia; by using test glasses—if the retinoscopic mirror has determined the condition to be hyperopia, the movement of course being with the mirror, now by placing a + 1. D. spherical lens over the eye if the movement still be present we know that all the hyperopia is not neutralized; if now with a

2. D. spherical, both in the vertical and the horizontal meridians the movements cease, we may conclude that the condition is one of general hypermetropia of two dioptres. If, however, the movements in the vertical seem to cease, but in the horizontal still persist, and with a 3. D. in the horizontal cease while in the vertical are reversed and move against the mirror, the case is one of compound hyperopic astigmatism, of two dioptres hyperopia, and one dioptre astigmatism; the same method of procedure would apply to all conditions, always remembering that a hyperopia whether simple or astigmatic or in combination causes a movement with the mirror at all times, and that a myopia whether simple or astigmatic, or the combination of the two, always produces a reversal of the movements, against the mirror, at all times. There are of course cases of spasm of the ciliary where a temporary myopia is produced; this is not very frequent and usually met with in children and young persons. It must be mentioned simply to be kept in mind as a condition occasionally met with; in relieving these cases a cyclophegic is necessary.

The relative frequency of these mentioned errors of refraction may be of interest. Most people are hyperopic, and of these most cases have astigmatism associated. Myopia at birth is rare, though in young children occasionally met with. It is, however, after reaching the age of from ten to fourteen that many cases of myopia show themselves; here the condition is probably an acquired one, from over or improper use of the eyes; astigmatism is fairly common with myopia.

In concluding this paper it must be mentioned that while the retinoscope is an accurate test of the refractive conditions of the eye, with but slight exception, and while it is possible to measure the amount of error with the mirror and the plus and minus neutralizing lenses, that it must never be the sole test for the prescribing of correcting lenses, but must be taken in combination with the other tests, the ophthalmoscope, the ophthalmometer, the retinoscope and the test lenses; if those four tests agree, glasses may be prescribed with a certainty of affording relief of asthenopia.

The purpose of the paper is to draw attention to the fact that in retinoscopy the physician who is not a specialist in ocular conditions has a test that with some patience and more practice can acquire skill enough to determine the refractive error present in the case in examination; by so doing he can always save the patient the discomfort and in many instances the markful results caused by the wearing of improperly and ignorantly fitted glasses.

Medicine may not be an exact science nor does the worker in the medical sciences know it all; there are some things that have come to us through work and study. Of these retinoscopy should be only in the hands of those qualified by study and knowledge of the anatomy and physiology of the eye; it should not be possible for any to ignorantly vaunt themselves as having the required knowledge and skill to determine and correct the errors of refractions of so delicately a formed and functioned organ of the body as the eye. Many and glaring are the so common examples; the jeweller thinks himself competent and so puts "Eyes tested here" on his show window—the "refracting optician" abounds; in most of our large cities his signs are sure to catch the eyes of passersby; the positiveness of his statements are alluring; his ignorance and lack of skill, however, are frequently laid bare in the office of the oculist.

The purpose of the paper is to present to the general practitioner a test for the refractive conditions of the eyes; also to call attention to its abuse in the hands of those unskilled and ignorant; a class outside of the medical profession that seems to be on the increase.

XXXI. THE PHYSICIAN AND THE OPHTHALMOSCOPE.

By FRANCIS VALK, M. D.

NEW YORK

At the last meeting of the State Medical Society held in Albany, our retiring President, Dr. Henry L. Elsner, in his closing address "A Modern Diagnosis" used these words, "An instrument of precision too little employed by the general practitioners is the ophthalmoscope, for the introduction of which the memory of Von Helmholz will always be revered."—"It gives information regarding the existence and nature of pathological conditions elsewhere than in the eye which is often obtainable in no other way." As I listened to his argument on the use of the ophthalmoscope I asked myself the question, How many physicians know how to use this extremely valuable instrument? Why do they not use it? Can its use be so demonstrated to them that they may use it?

These are questions to be answered partly by the physicians themselves but the last must be answered by one somewhat familiar with the use of the ophthalmoscope and with the advantages it may present to the physician in the diagnosis of many conditions that arise in general practice, either that it may elucidate some unexplained phenomena or confirm some previously suspected conditions.

It goes without saying that every physician should make himself as fully competent as possible in the diagnosis of all general diseases, with their complications and their sequela. In the use of all other instrumental aids, as the stethoscope, etc., the physician does inform himself as thoroughly as possible, yet in the use of this instrument, one of the best for the objective diagnosis of disease, he seems to have left it in the hands of the specialist; depending possibly on a consultation or passing the matter to one side depending solely on his other objective and subjective signs.

There are few instruments in the hands of the physicians that can give as useful information in the diagnosis and prognosis of disease as that of the ophthalmoscope nor is there one that can be relied upon so implicitly. The findings of the ophthalmoscope are so obvious and correct that they leave no doubt as to the condition of the fundus and the systematic conditions that these findings may indicate. Furthermore, as Gowers well says, "We examine the artery to its termination and the commencement of a vein with the blood circulating in each; the termination of a most important nerve which, from its close proximity to the brain, undergoes significant changes in various disease conditions of the brain and in affections of all other parts of the nervous system; and a nervous structure, the retina and a vascular structure, the choroid, which also suffer in a peculiar way in many general diseases." Is not this matter important enough for a careful consideration?

I do not expect that the general practitioner will ever become so far proficient as to readily detect all the most delicate changes that are sometimes presented by this beautiful system of vessels, nerves and tissues of the eyeball, yet I do believe that with reasonable experience in the use of this instrument the physician may so perfect himself as to recognize the most important changes in these delicate structures of the eyeball indicative of or associated with some systemic disease. What can be more useful than the picture presented by the appearance of the termination of the optic nerve in any obscure disease of the brain or that of the retinal vessels and the retina itself in diseases of the kidney? Do we not know of cases of Bright's in which, the first correct diagnosis has been made when the eyes have been examined with the ophthalmoscope? Tuberculosis may be diagnosed or at least confirmed with the opthalmoscope by the appearance of the fundus, as it shows the characteristic tubercles in the choroid, and in doubtful cases of acute miliary tuberculosis may confirm the diagnosis. It is not always possible to refer these cases to those who are engaged in this special field of medicine and one

who can use this instrument—even in a comparatively limited manner—will be much more than compensated from the instruction and the information it will give him. The brief time it may require to learn the method of its use in a practical way is not very great.

How often have I heard the answer to the question, Have you an ophthalmoscope? Yes, but I never use it; but why? Why does the physician possess this instrument and yet not use it? I think I can answer this question in this way. Because when he commenced the study of ophthalmoscopy in the text-books he found it presented so many details and so many complications that the instrument was soon laid aside, simply to be remembered in the possession of its owner but not to be used. It is my hope that this subject may be presented in a very simple manner, by one partially familiar with its use and advantages, which is the object of this paper, and I would present it by the common sense method, so that it will be useful to the physician and leave the more complicated uses of the instrument to the specialist.

Before proceeding further let me describe the instrument. What is it? It is simply an apparatus by which we may place the examiner's eye in the path of the rays of light as they come from the background of the examined eye when illuminated by reflected light.

To see an object we must have the rays of light pass into our eyes in certain directions, either parallel or divergent. Now the rays coming from an examined eye are either parallel, divergent or convergent. To the second condition—divergent rays—our eyes are readily adjusted and we have a perfect image on the retina of the examiner, the fundus being easily seen. The parallel and convergent rays cannot be focussed on the retina—and here is one of the trials of the beginner—except under special conditions, but they can be easily made divergent and so become useful for the vision of the examiner. Then our instrument is a mirror to illuminate the examined eye and a disk of lenses to so bend the emerging rays that they may focus on the retina of the examiner.

To illuminate the eye we require a good steady light—the Argand burner with oil or gas is the best—a slightly concave mirror to concentrate and reflect the light and a handle to direct the reflected rays, within the eye-ball, directly through the pupillary space. If we now place the examiner's eye in the center of the rays coming from the examined eye—that is behind the aperture—as the retina is illuminated, he will be able to see the details of the fundus if the out-coming rays are divergent. If not divergent we cannot see the fundus clearly and we must

make them so by placing suitable glasses behind the aperture. It can be readily understood that if the examiner's eye receives these rays and can focus them on the retina they must there form a picture that will correspond with the object from which they proceed. In other simple words, the ophthalmoscope enables us to look into the eyes of our patients and to see the condition of the eye-ground, either in health or disease. Such is the simple purpose of the ophthalmoscope, seemingly very easy but I will point out the difficulty that may arise and then show the usefulness of the instrument.

It has been fifty years or more since the ophthalmoscope was first used to see the interior of the eye but to Von Helmholz we owe all reverence for his introduction of this instrument in 1851 and those of to-day are the same in principle, only differing in the mechanical parts, as when the instrument was first used. I do not propose to enter into the subject of the estimation of the refraction of the eye but only how to see the background or retinal field; what it looks like and what are the essential changes that we should be able to see, to know and to appreciate. Now the text-books tell us to look through the aperture of the ophthalmoscope as if we were looking towards infinity or in the distance with the accommodation at rest, etc. This is all very true but it requires a vast amount of practise to master just that; no beginner can do it nor is it essential and moreover it is only useful to the specialist. I shall then try to inform you as to the use of the instrument as needed in the hands of the general practitioner. Let us accept the statement that one is utterly ignorant of the method of using the ophthalmoscope but wishes to understand its use in the diagnosis and prognosis of disease, states of the general system. How then shall he use the ophthalmoscope so that he may gain this practical knowledge? Place the light—let me say at once that too bright a light is not necessary for the appreciation of the most delicate changes at the fundus—behind the patient, about one foot distant from the eye, on a level with it and to the right or left according to the eye to be examined; seated now on the side of the eye we wish to examine, the ophthalmoscope is placed before the corresponding eye of the examiner and he looks through the small opening or aperture. If possible, it is best to keep both eyes open, but for the purpose we have in view, one may close the eye not used if by so doing it will assist us in the examination. Now reflect the light from the lamp or gas-burner into the patient's eye and we find the pupil changes from its natural blackness to that of a bright orange red. If not so we must proceed to a more detailed examination of the anterior part of the eye as the cornea and

lens. To do this, we place the lens marked + 6. in the disk, at the aperture and we move nearer the patient until we are within the focal distance of the lens—about five or six inches—when we will see the anterior part of the eye magnified and can readily detect any opacity or changes that may appear on the cornea or lens, showing generally as black spots on the bright orange red color of the pupillary space. This examination may indicate incipient cataract, iritic deposits on the capsule or if there is complete absence of the pupillary reflex some very serious condition of the interior of the eye-ball. To resume, if this portion of the eye is found to be perfectly clear we now proceed to examine the deeper parts or fundus. Removing the convex lens and again looking through the aperture, with the reflected light directed into the pupillary space, we now attempt to see the background or fundus of the eye, in other words, the nerve, retina, vessels, the choroid, etc. But just here comes the great difficulty which all examiners meet in their first attempts to examine the eye. This is due to the direction of the rays of light coming out from the examined eye and the inability of the examiner to control the action of his own accommodation. We make an effort to look at something very close to us on account of the proximity of the patient's eye and so we exert a certain amount of accommodative power, preventing the formation of the image on our own retina, with blurred vision. To obviate this difficulty, so common to beginners, we must place at the aperture one of the concave lenses marked with red figures on the disk. This will neutralize the action of our own accommodation, will make the rays of light divergent and then the details of the fundus will rapidly come to view.

Now having mastered this instrument to the point where you can begin to see fundus you must know what you see and what to look for, both in health and disease, so that you recognize such pathological conditions of the fundus that may point to some systemic condition. If you will attempt to make this examination on the eyes of your patients that have no eye troubles, young people with large pupils, you will soon appreciate what is the appearance of the normal fundus with the arteries, veins and nerve. With this mental picture before us we can readily see any pathological changes that may have taken place. Before proceeding further, let me state, inasmuch as the pupillary area is not always the same in every eye and when very small it is very difficult, even for an expert, to see anything except the red reflex, then you are justified in using a weak mydriatic to dilate the pupil. Atropine will not do for this purpose as it impairs the sight too long.

Cocaine, four per cent. or homatropine, one-fifth of one per cent. will be the best, dropped in the eye about twenty minutes before we are ready to begin the examination. Either one of these drugs will dilate the pupil in a short time and will make your examination much easier, while the effects of the mydriatic will pass off in about twenty-four hours.

All these conditions now being at our command the next question arises: What is the normal appearance of the fundus or background of the eye, and in the words of Haab "I cannot too strongly advise the beginner to study the normal eye as often and as thoroughly as possible."

We first note the color of the eye-ground; this will appear very different even in perfect health, according to the individual, as blonde or brunette. Brunettes generally present a much darker eye-ground than blondes. This difference is chiefly due to the amount of pigment in the retinal layer, the pigment epithelium. This pigment is also in the choroidal layer showing as dark spaces between the choroidal vessels, and in the negro the dark spaces and the numerous choroidal vessels are very often seen, while in the albino, with little or no pigment, the vessels of the choroid will appear red on an almost white back-ground. In the region of the macula or yellow spot—the position of most acute vision—always found outwards, toward the temples from the nerve entrance, we find the pigment color darker and in many cases a small spot in the centre. This pigment is also seen about the entrance of the optic nerve, forming the choroidal ring, dark masses irregularly placed but seldom if ever extending completely around the nerve. The nerve entrance is quickly seen, being so prominent, as a large round space, apparently in the centre of the eye-ground. It presents a grayish white color, seemingly semi-transparent, and the central part whiter from the dipping inward of the central nerve fibres. If the central part is very large and deep, the lamina cribrisa may be seen as a large white network in the nerve entrance. This nerve entrance or papilla may be round or slightly oval in any direction, and in a condition of health its surface does not extend beyond the plane of the retina. The retina itself in health cannot be seen except in some cases by the most practised eye. The general appearance of this eye-ground presents to the examiner's eye a peculiar appearance as of a very small irregularly figured carpet, somewhat like a mosaic of small light and dark spots; this is called the *stippling of the retina.* This is very important and should be carefully studied as its presence or absence must indicate the condition of the retina. The slightest changes being shown by the blurring of this delicate network of pigment, indicating some

pathological condition either in the eye itself or the parts behind it. The arteries of the eye-ground, consist of the branches of the central artery of the retina, which pass out from the nerve and separating into smaller arteries extending outward over the entire retinal field to the anterior part of the eye-ball; similarly, the veins of the eye-ground have the same arrangement, are darker in color and empty into the central vein of the retina. We can recognize the arteries by their lighter color and the white line that runs along the center of each vessel while the veins present a much darker color, the line along the center not being so prominent. All these vessels have a straight course, being slightly curved and they can be distinctly traced to the periphery of the retina. Any irregularity in their course or blurring of their outlines is probably due to some retinal changes. Pulsation in the veins, on or near the nerve entrance may be readily seen but have no special significance, but are serious if seen in the arteries. The diagnosis of this condition is very difficult and would carry us beyond the scope of this paper. For a more complete description of the normal eye-ground I must refer you to the many textbooks on this subject but I have described the most important details of the appearance of the normal fundus as seen with the ophthalmoscope in order that we may become familiar with this picture from the examination of the eye in health so as to appreciate the changes that take place in disease as shown by the retina, nerve and vessels.

Because of their frequency and their importance in the diagnosis of disease, there are two conditions of the fundus of the eye that will always have special interest to the medical profession. I refer to retinitis, in its many manifestations and what is commonly called "choked disk." Retinitis, inflammation of the retina, covers a vast variety of changes in the back part of the eye, readily seen by the expert with the ophthalmoscope. The general practitioner cannot see all the minute changes, yet with reasonable care he can appreciate the important changes that may indicate some local or systemic disease if he becomes familiar with the appearance of the normal fundus. Now inflammation of the retina becomes the most important change because it is frequently present in so many other systemic diseases that may be first noticed from the complaints of failing vision, as if there was a veil or blur over the page when reading. According to Jacobson, (*New York Medical Journal*, July 1901) inflammation of the retina occurs in ten per cent. of the cases of Bright's disease and is known as albuminuric retinitis. Its significance is very important, as it is well known that when these changes take place in the retina the possible duration of life is seldom more than two years.

I made the diagnosis of Bright's disease with the ophthalscope in a case of supposed hysterical amblyopia and in three weeks from that time the patient, a young girl, passed away. The importance of this examination must be obvious to all and if we have the pupil dilated with any brief mydriatic, and retinitis is present, then, as we look into the eye with the ophthalmoscope, we will see a great difference in the appearance of the retina from that of the normal. The edges of the disk will not be clearly seen; the veins will be dark, tortuous and dilated; the arteries partly covered by the swollen retina, cannot be followed to the periphery and seem to be covered by white masses, like clouds, that are partly due to exudation and œdema of the retina. Then we find various dark, irregular spots, small and large, scattered about the retinal field, due to hemorrhages from rupture of some of the small vessels of the retina. In some cases these hemorrhages may be very large and in others very few or even entirely absent. White patches about the retina are frequently seen, in which we note that the edges are not very clearly marked but shade off into the retina and the small blood vessels seem to pass through them. Fatty degeneration of the retina is also present and shows itself as a peculiar stellate appearance in the region of the macula or yellow spot. These minute spots are arranged in rows, radiating outward from the center of the macula region, not always perfect, as they may be absent on one or the other side, but when present with the other appearances are almost positive evidence of kidney disease. Knapp states that this degeneration of the retina may be seen in tumor of the brain but I have not been able to see any cases yet that were not associated with albumin in the urine. The swelling of the retina may extend so as to involve the nerve-head or disk with blurring of its outline and reddening of its surface; in advanced cases this swelling may resemble "choked disk" or papillitis but the presence of the stellate figure in the region of the macula, the white patches and the hemorrhages will confirm the diagnosis of Bright's.

I do not know of any more important use of the ophthalmoscope to the practitioner of medicine than the diagnosis of the condition of albuminuric retinitis. The changes that may be shown in the early stages of this disease may present a slight congestion and tortuousity of the vessels with blurring of the retina and the edges of the disk; this condition associated with diminished vision would lead us to suspect diseases of the kidneys and at a time when it may be amenable to treatment, but when we have the characteristic picture as presented then we can only say that the prognosis is very grave with probably a fatal result in less

than two years. It has been claimed by Pooley that the ophthalmoscope will assist us in the diagnosis of the albuminuria of pregnancy and if present is an indication for premature delivery but I have had no experience with these cases and without other signs should prefer to wait.

Choked disk or papillitis: under this heading we may consider most of the inflammatory changes of the optic nerve or papilla. The ophthalmoscope appearances of the condition may be readily understood when we consider how prominent the disk is, the normal appearance of the disk, its distinct circular outline and the branching outward of the vessels from the center. Now in choked disk we have an almost complete disappearance of all the characters of the normal disk and an examination there presents a distinct swelling of all the nerve fibres as they seem to stand out in the vitreous body, covering over parts of the blood-vessels and then shading off, as it were, to an almost normal retina. Such is the simple picture of the choked disk and the appearance it usually presents at the ophthalmoscopic examination; but in some severe cases the swelling of the nerve-head is much more extensive, the vessels very tortuous, and extensive hemorrhages throughout the entire retinal field. To the expert with the ophthalmoscope this swelling or extension of the nerve-fibres into the vitreous may be measured, giving about .34 m.m. to each dioptre of refraction, so that he can note any increase or decrease of the elevation. Is not the diagnosis of optic neuritis or choked disk very important in the practise of medicine as it is one of the important signs of an abscess or tumor of the brain, a sinus thrombosis and other inflammatory systemic conditions?

Optic Nerve Atrophy: In tabes and other conditions where we have a diminunition of the visual power, dim sight, the opposite condition to that of choked disk is found. Here we have all the details of the fundus and the nerve head very clearly shown but the natural soft color of the disk has changed to a hard, white or slightly bluish color. In tabetic atrophy the optic nerve has a pearl gray color. The edges of the disk are very sharp and clean cut, the lamina cribrosa shows as an interlacing of the nerve-head and all the vessels appear much smaller than normal. In the gray atrophy of tabes the prognosis, as to any restoration of sight, is very serious as they generally pass on to complete blindness.

Amblyopia or blurred vision, due to atrophy of the optic nerves as shown by the ophthalmoscope, and caused by the abuse of alcohol and tobacco is probably more amenable to treatment and the prognosis is not so serious. This condition is more apt to affect the central nerve fibres or those supplying the macula

region, and this is shown by a peculiar whiteness of the disk in the lower outer quadrant. Here we find the evidence of atrophy from the central scotomata or blindness in the centre of the field of vision; and conversely, in the tabetic atrophy the blindness commences in the outer part of the field and extends towards the centre. I have some doubts about the atrophy of the nerve being caused by tobacco alone but when we have the condition of alcohol and tobacco used in excess and when the use of toxic drugs has been abused in early life then I think they may bring about an atrophy of the optic nerve fibres. This may possibly be controlled by the stoppage of these pernicious habits, the use of strychnia by hypodermic injection and, in some cases, the iodides. I have never seen complete blindness from this *amblyopia ex abusa.*

Choroiditis: Changes in the appearance of the fundus of the eye is frequently seen as large white plaques with spots of pigment on the edges, and scattered about the retinal field are many pigment deposits looking very much like bone corpuscles. The retinal vessels are seen to pass over these white spaces without any deviation showing that they are not involved in the process. This appearance is due to an inflammation of the choroid that may have been active but is now the result of a previous inflammation. In the acute stages of exudative choroiditis we have an infiltration of the choroid with exudation that hides the retina and through which the retinal vessels seem to pass. As resorption takes place the exudate disappears and the choroid presents these atrophic spots. Suppurative choroiditis is very serious and is generally metastatic. Bull states that he has seen several cases of infectious choroiditis, due to *la grippe,* in which the ophthalmoscope gave a most startling picture with ultimate destruction of the sight. This septic choroiditis is also met with in typhoid and other fevers, in pyæmia, in meningitis and in puerperal septicæmia. The amaurotic cat's eye or pseudo-gliomata is a mild form of suppurative inflammation of the choroid, showing as a whitish or yellow reflex, deep in the eye, the pupil dilated and the tension of the eye decreased. This condition must not be mistaken for a true glioma of the retina that is always associated with increased tension of the eye-ball.

Embolism of the central artery of the retina may be the first indication of an endocarditis. This is well shown by the whitish color of the retina, about the centre, probably due to an exudation. The vessels are indistinct and in the centre of the macula a cherry red spot is seen. This is a typical picture and is only seen, as far as I know, in this condition and in the peculiar disease known as amaurotic family idiocy.

Hemorrhage into the retina is frequently seen and if without

any signs of inflammation would indicate an increased blood pressure, disease of the retinal arterial walls or some morbid states of the blood. In old people it shows a weakness of the arteries. These hemorrhages will show with the ophthalmoscope as dark red spots of various sizes and in any parts on the retinal field, some of them very minute and others very large. In diabetic retinitis very small hemorrhages may be seen but generally only in one eye; and also in the diagnosis between chlorosis and pernicious anæmia as these hemorrhages are not seen in the first named disease but are frequent in the latter.

Glaucoma with increased tension of the eye-ball and diminished vision. The ophthalmoscope will show a cupping or pushing backward of the entire disk so that the blood vessels tip sharply at the edges and appear indistinct in the centre. This condition is generally seen quickly with the ophthalmoscope and should not be mistaken for atrophic changes in the disk.

The use of the ophthalmoscope in the diagnosis of all the many changes that occur at the fundus would carry us far beyond the scope of this paper so I have only considered those most useful to the physician.

I do not expect that all physicians will become so proficient in the use of this instrument, from a careful study of this paper, as to use the ophthalmoscope with all the skill of an expert. It requires constant application and experience even to master these details but on the other hand, careful study and practise of the method I have endeavored to convey in this paper will soon enable one to see, to know and to appreciate the appearance of the healthy back-ground or fundus of the eye and seeing and knowing this we may readily appreciate any changes that have taken place, that is to say: The pathological condition of the retina and its adjacent structures and the systemic disease that is the prime cause.

It is impossible to describe the many conditions of the eye-ground that may be seen with the ophthalmoscope, nor is it necessary in a paper of this import. The diagnosis of all the many changes that may be of pathological interest must come from one who is fully competent to use the instrument in all the various conditions of the eye and the correction of its refraction, but I trust that I may have made some suggestions that may be of service to the physician in his work. In closing I again turn to the words of Elsner, "We do not contend that the general practitioner must of necessity become an expert in the use of the ophthalmoscope but we believe that he must depend on its revelations, if we would early recognize certain diseases of the brain, arterial degeneration and renal complications."

XXXII. AN OPERATION FOR CICATRICIAL CONTRACTURES OF THE UPPER EXTREMITIES.

By A. H. TRAVER, M. D.

ALBANY

It would be a tiresome task for one to review all the numerous operations that have been performed for the purpose of curing cicatricial contractures, so I will proceed at once to the description of my case:

Case.—L. W., a male, aged seven.

History. As the patient is an orphan, it is impossible to obtain a very complete history. A friend states that when the patient was four years old, while pouring oil on the fire, he sustained a severe burn of the right arm. This was followed by drawing up of the arm. Nothing is known of the treatment carried out at the time of the injury. In October, 1900, he was operated upon by another surgeon. This operation consisted in making transverse incisions five-tenths centimeter apart through the cicatrix involving the elbow, and the application of an anterior straight splint. No attempt was made to straighten the wrist. The splint was worn about ten weeks, but after its removal the arm became flexed again to practically the same position as before the operation. The patient came under my care at the Child's Hospital in February, 1902.

Examination. Patient was poorly nourished, very anemic and exceedingly nervous. The elbow was flexed to an angle of 70°, the wrist to a right angle. The cicatrix extended from the shoulder to the metacarpophalangeal articulation. The entire circumference of the arm was involved, except a strip two centimeters wide along its inner surface. The cicatrices at elbow and wrist were firmly contracted. Through the cicatrix at the elbow were seen the transverse cicatrices of the previous operation. There was an ulcer one centimeter in diameter over the olecranon that had been there for over a year. Owing to the overflexion of the wrist, caused by the cicatricial contracture, the scaphoid and semilunar bones were dislocated and could be moved freely about beneath the skin. The arm was practically useless. There was slight motion at the elbow, but none in the wrist. The patient was unable to feed himself or even touch his face, except with the back of his wrist. If he tried to raise any weight it caused severe pain in the wrist.

I began treatment of the patient, feeling that the arm was of no use in its present condition and hoping that something might be done to place him in a condition to earn a livelihood.

Treatment. First operation was performed February 8, 1902. A band of the cicatricial tissue ten centimeters wide surrounding the elbow was dissected away, leaving a small strip of fairly normal skin two centimeters wide along the inner surface of the arm. This permitted the arm to be placed in nearly complete extension—complete extension, however, being prevented by the contracted biceps muscle. It was thought advisable not

Before operation.

After operation.

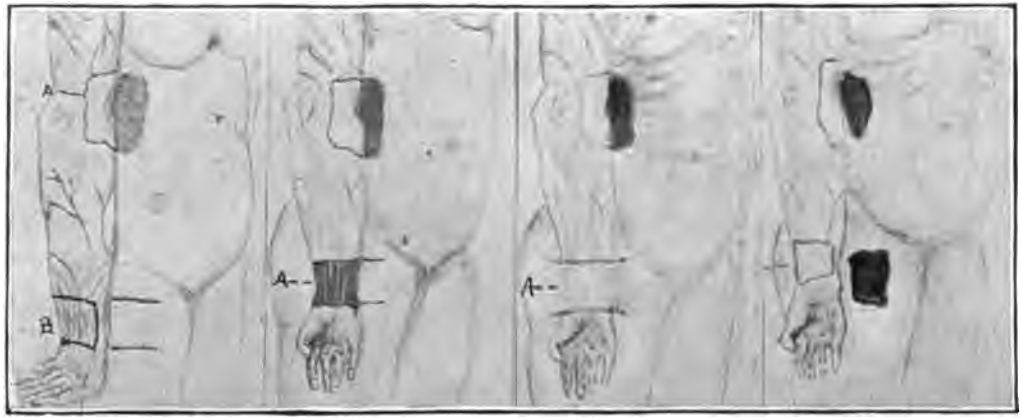

FIG: 2.—(1) a, flap separated from chest and sutured to arm; b, arm to be denuded. (2) a, cicatrix dissected away. (3) a, arm beneath skin of hip. (4) arm free from hip.

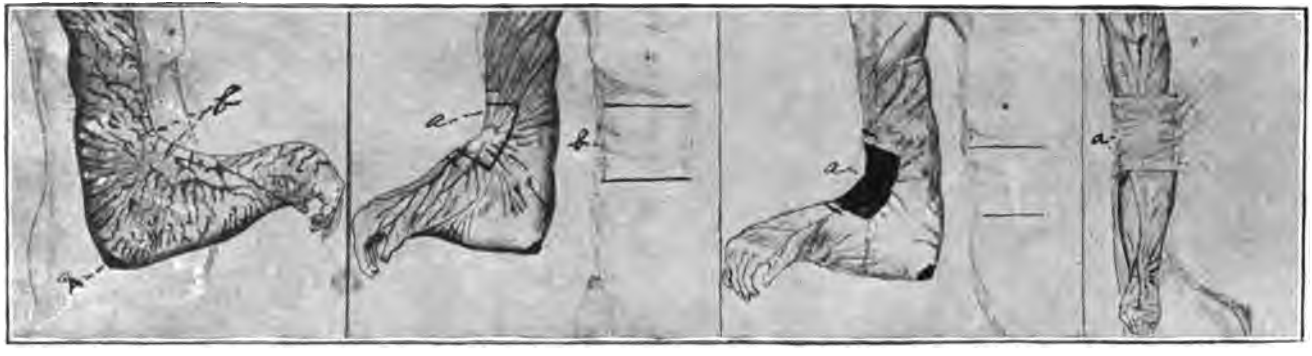

FIG. 1.—(1) a, ulcerated surface; b, scars of previous operation. (2) a, area to be denuded; b, flap to be raised. (3) a, area denuded. (4) a, arm beneath skin of chest. [Courtesy of *American Medicine*.]

to interfere with this muscle, as the slight flexion would be of little hindrance to him in performing his work in after years. I then raised from the side of the chest a flap of skin twelve centimeters broad by twenty centimeters long, the ends of this flap being left intact to keep up the circulation. The skin on either side of the chest from which the flap had been raised was dissected up and by means of tension sutures brought together as much as possible, thus closing about one-half of the gap. The arm was then drawn through beneath this flap, thus covering the surface on the arm, from which the cicatrix had been dissected away. A strip of gauze was drawn through beneath the arm to prevent its growing fast to the chest. Dressings were applied and arm and chest put up in plaster-of-paris. Operation was followed with but slight fever and patient complained of but little pain. Dressings were left undisturbed for eighteen days, at which time the flap had become firmly adherent to the arm.

The second operation was performed February 26, 1902. The ends of the flap were severed from chest, brought about the arm, and sutured in place, thus completely covering the denuded area on the arm. In bringing the flap about the arm, we found we had a small piece of skin in excess of that required, so this was utilized as skin grafts on the chest. Sterile dressings were applied.

The third operation was performed May 1, 1902. A band of cicatricial tissue seven centimeters wide surrounding the wrist was dissected away, leaving a strip two centimeters wide over the extensor surface. The tendons of the flexor carpi ulnaris and radialis were so tense that it was necessary to lengthen them two centimeters. Even now, after removal of cicatrix and lengthening of the tendons, the wrist cannot be fully straightened owing to the deformed condition of the carpal bones. A flap of skin ten centimeters wide was raised from the right thigh and the hand slipped beneath. This flap was sutured to the wrist. Sterile dressings and plaster-of paris were applied. This dressing was left on for fourteen days.

The fourth operation was now performed. The flap was severed from the thigh and brought about the wrist in the same manner as at the elbow. Skin grafts were removed from the opposite thigh and applied to the surface from which the flap had been taken.

On June 11, 1902, the patient went to the Orphan Asylum. The elbow, wrist and chest were healed. There was still a slight area on the thigh unhealed. The elbow was practically straight, but the wrist was still slightly flexed.

Several times during the summer I have applied plaster-of-paris dressings to the wrist, holding it in as extended a position as possible until the plaster hardened, hoping thereby to get the carpal bones to return to their normal position. Each time there has been an improvement, so that now the wrist is straight, as can be seen by the photograph, but complete extension is impossible.

In the beginning the arm and wrist were drawn in complete flexion and were useless to the patient. He would doubtless never have been able to have sustained himself. Now, although the arm is not as shapely as the other, yet it is nearly straight and is very useful. He is able to play

games, can feed himself, and is learning to write. He can raise ten pounds weight with this hand and will be able to earn a living.

This manner of treating cicatricial contractures of the elbow and wrist may have been previously carried out, but I am unable to find any similar case described in literature. The only case in any way like this that I can find described is that of Schoeder's in the *American Journal of Medical Sciences,* October, 1900. (This article had not been seen by me until after completion of my case.) He raised a flap of skin from the thigh to cover the palm of the hand. In this article he states that in order that the flap may retain its vitality there must be no tension on it. In my case tension was of necessity put on the flaps by the passage of the arm beneath them, but it did not interfere in the least with their vitality.

XXXIII. ACUTE OSTEOMYELITIS.

By ARTHUR. W. BOOTH, M. D.

ELMIRA

The rapid increase of knowledge pertaining to surgical pathology has created a necessity for many changes in surgical nomenclature to render it consistent with our broader conceptions of the morbid processes. Thus, the earlier writers described several suppurative bone diseases and employed such terms as acute ostitis, endostitis, medulitis, periostitis and parostitis, all terms more or less misleading and incomplete in that they describe the progressive steps of a single disease, viz.: Acute suppuration of bone. It would appear, then, advisable to discard these names entirely, and apply the more comprehensive term,—acute osteomyelitis.

This disease furnishes the greater number of necrosis cases brought to the surgical clinics, and, in most instances, the fact that extensive necrosis has occurred is a reproach to modern medicine. It should not be looked upon as a specific disease, being nothing more than an abscess in a compact tissue. This disease is far from rare, and has a mortality greater than appendicitis, or typhoid fever, yet is but poorly understood by the general practitioner and is frequently overlooked until irreparable damage or even death have occurred. Mr. Holmes has said "acute osteomyelitis is more frequently recognized at the post-mortem examination than at the bedside."

Aside from those cases due to known sources of infection such as are found in amputation stumps, gunshot wounds and compound fractures, we have that class sometimes called "spontaneous,"—cases of unknown origin. The disease may manifest itself by multiple infections, but usually a single bone is affected and there appears to be a predilection for the long bones, especially the femur, tibia and humerus. It is distinctly confined to childhood and youth, during the stage of most active bone growth. In nearly every case there can be elicited a history of traumatism to the affected bone. This injury may be ever so slight and frequently is disregarded as a factor in the case, yet it must have produced some alteration of the medullary circulation. Exposure to wet and cold after fatigue is regarded as an exciting cause. Ulmann produced experimental osteomyelitis by injecting pyogenic material into the veins, but the bones were not the seat of suppuration unless they had previously been subjected to some form of traumatism. The essential cause in all cases is some pus-producing micro-organism carried to the medullary substance by the circulation. This metastasis may have arisen from an insignificant surface wound. Kraske mentions the tonsil as the point of origin. Many attribute infection from the respiratory and alimentary tracts. The various fevers, notably typhoid and the exanthemata, may cause the disease both by their depleting effect and by their specific germs. A simple fracture may terminate in osteomyelitis when the fracture occurs during the course of suppuration in some distant portion of the body.

In studying the pathology of this disease, it is observed that the inflammatory process usually begins at the seat of most active bone growth,—near the epiphyses. The compact tissue is never primarily infected, and primary involvement of the periosteum is exceedingly rare. A glance at the anatomy of the blood paths show that the capillaries near the epiphyseal line and in the myeloid tissue are imperfectly developed, are devoid of proper vessel wall and, as Neumann has pointed out, their calibre is four times greater than that of the arterial branches that supply them. These peculiarities present favorable conditions for deposition of floating microbes or infected leucocytes. The intense phlegmonous inflammation following infection rapidly converts the medullary canal into a pus cavity. Extravasated blood and oil globules give a characteristic appearance to the pus. In some cases, however, very little pus is in evidence, the extravasated blood and thrombosis predominating. For an interval, the pus is confined to the bone canal from which it later spreads through the cancellous tissue out towards the periphery, and collects underneath the periosteum, causing extensive denudation, and ultimately it

invades the surrounding soft parts. Along with this pus formation, there occurs thrombosis of the veins and occlusion of the blood vessels, which are the immediate causes of the necrosis. The thrombi undergo liquifaction necrosis and thus establish pyæmia with secondary deposits to distant organs.

This disease is abrupt in onset, usually ushered in by a chill, followed by a constant high temperature, with exacerbations at night. Pulse is rapid and small, tongue soon becomes dry, the patient presents an anxious countenance and progresses on to the full picture of profound septic intoxication.

The local symptoms are severe pain, manifested early, of a boring and throbbing character, aggravated by the tension within the non-yielding bone. This pain is not restricted to the involved area, but may extend to the adjacent joint and greater part of the shaft, and may be "referred pain" through the nerve paths. Still, by careful palpation, it is possible to map out with fair accuracy a localized tenderness which corresponds to the area most actively involved. Loss of function appears early, the patient cannot move the limb and complains bitterly when it is moved, expressing a fear of breaking the bone. This fear is quite characteristic of the disease. Swelling and redness do not appear until the pus has reached the periosteum whereupon the presence of pus becomes unmistakable. Engorgement of the subcutaneous veins is usually present and indicates obstruction of the deeper veins.

Further local symptoms are:—Epiphyseal separation, spontaneous fracture, effusion and suppuration of adjacent joints, pus sinuses and bone necrosis. Not the least to be considered are the overshadowing symptoms of pyæmia in progressive cases, the meninges and lungs are frequently invaded and there may be fat embolism.

Acute osteomyelitis bears an unfortunate resemblance to acute rheumatism, especially if the joint has become involved. On account of this, a surgeon seldom has the opportunity to observe the case early. The swelling and redness, more or less localized over the pus collection, are sometimes mistaken for erysipelas, or phlegmonous cellulitis. The fulminant type runs its course so rapidly that the local symptoms appear insignificant, and a faulty diagnosis of typhoid fever is the result, or death ensues before the disease is recognized. There is no condition met with where it is more important, both to the physician and the patient, to arrive at an early and accurate diagnosis. Rheumatism is not, as a rule, restricted to one joint, the temperature is not so high nor so constant. In osteomyelitis, the general symptoms are all out of proportion to the local manifestations in the bone or ad-

jacent joint. Erysipelas and cellulitis present redness and swelling at the onset, whereas, in osteomyelitis these symptoms, due to pus in the periosteal sheath, are preceded by an interval of temperature and obscure pain. Typhoid fever has usually quite characteristic prodromes and the Widal test would help clear up the situation. In typhoid and rheumatic fevers, we do not find an increased leucocytosis which is always present in osteomyelitis. Under favorable conditions, the Roentgen rays might be utilized in detecting a collection of pus.

As to treatment, there is no middle ground. Expectant measures do not even deserve mention. Having made the diagnosis, the rule should be, open early and open thoroughly. One of the saddest errors is, having exposed the periosteum and found pus there, to rest content that the operation is complete. Primary infective periostitis is exceedingly rare, we should proceed further and chisel through to the medullary canal, feeling confident that pus will be found there. This cavity should be well curetted, disinfected, and packed with gauze for drainage. Some advocate exposure of the entire length of the shaft down to the opposite epiphysis, removing a considerable portion of the cancellous tissue and leaving only an outer shell, regeneration of bone is reasonably assured. Others rely upon openings at either end of the bone and pass gauze through these so as to drain the entire bone. If the periosteum has been invaded by pus, some cortical necrosis must be expected. When operative interference has been early, the gravity of the situation is much lessened; immediately after the evacuation of pus, the general symptoms of septicæmia subside with surprising rapidity. From an extremely anxious outlook, the case takes on the aspect of hopefulness. Some degree of necrosis and deformity may follow, but life has been preserved, and the subsequent damage can be met under better general conditions and more convenient circumstances.

XXXIV. PRIMARY CARCINOMA OF THE VERMIFORM APPENDIX, WITH A REPORT OF THREE CASES

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The exceedingly infrequent occurrence of primary malignant neoplasms of the vermiform appendix has long been a subject of comment among surgeons and pathologists. In the voluminous literature devoted to diseases of the appendix there are but comparatively few references to malignant diseases of this organ, and some of these are decidedly unauthentic. This appears to be especially true of the earlier cases reported. In order that some idea may be gained as to the accuracy of these observations, the writer has taken the trouble to look up the original reports, where such exist, of practically all the cases in the literature.

The first reference to carcinoma of the appendix is a case reported by Merling[1] in 1838. The report was of the findings at an autopsy upon a case which had died of general peritonitis. The wall of the appendix was thickened, scirrhous, and of a grayish-brown color. Furthermore, it appeared to be occupied by several small hard tumors. Near the attachment to the cæcum the appendix presented a small ragged opening the size of a pea, through which the intestinal contents had escaped into the peritoneal cavity. Although assumed to be a case of primary carcinoma of the appendix, no mention was made of a microscopical examination, and it would appear to be much more probable that it was a case of perforative appendicitis.

The next mention of primary carcinoma of the appendix was a case reported by Prus,[2] in 1865, of a woman seventy-five years of age who died with symptoms of general peritonitis. At autopsy the peritonitis was found to have originated from a large perforation of the wall of the appendix. The mucous and muscular walls of the appendix were thickened. From the base of the ulceration about the perforation there sprang a small, soft sessile tumor, which on section showed some hæmorrhage. No mention was made of a microscopical examination, and it would also appear that this case was not one of primary carcinoma of the appendix.

The first important article upon the subject was that of Rokitansky,[3] who in 1867 reported four cases of what he believed to be primary carcinoma of the appendix. He called attention to the fact that prior to that time carcinomatous degeneration of the

appendix had been scarcely recognized among the diseases of that organ.

The first case, which was observed in 1847, was that of an individual eighty-two years of age who died of pulmonary disease. At autopsy the appendix was found to be transformed into a sac six inches in length and two inches in diameter. It was situated to the outer and posterior side of the cæcum, to which it was densely adherent, and into the lumen of which it had caused a projection. The walls of the appendix appeared fibrous in character and the cæcal orifice of the appendix was obliterated. The sac which represented the degenerated appendix contained a yellowish white, opaque, gelatinous mass, which was streaked with blood. The wall of the sac consisted of fibrous tissue which had completely replaced the mucous and muscular coats, and upon the inner surface presented an areolated, reticulated appearance, the reticulæ traversing the gelatinous material and dividing it more or less into compartments.

The second case, observed in 1854, was that of an individual sixty-eight years of age who died of pneumonia. The appendix was two inches in length, and the distal two-thirds was transformed into a spindle-shaped fibrous sac about the size of a pigeon's egg, which contained a yellowish gelatinous material traversed by fine reticulated processes which took their origin from the wall of the sac.

The third case, observed in 1866, was that of an individual seventy years of age who died of heart disease. The appendix was four inches in length, and the distal, 2.9 inches, was transformed into a sac distended with a gray gelatinous material. This portion of the appendix was about one inch in diameter, and the wall, which was one-tenth of an inch in thickness, was of a fibrous character. The entire surface of this sac was smooth in some places, while in others it was irregular, and from it numerous delicate processes ran in different directions through the gelatinous material.

The fourth case, observed in 1866, was that of an individual thirty-eight years of age who died of pulmonary and intestinal tuberculosis. The appendix was 1.9 inches in length and consisted of two compartments. The distal, lined by a smooth mucosa, contained a grayish gelantinous material, while the proximal compartment was distended with a gelatinous material traversed by numerous delicate strands of tissue, which appeared to orginate from the fibrous wall of the compartment. The walls of the appendix in this region appeared to be transformed into fibrous tissue.

Rokitansky concludes that this disease of the appendix con-

sists of a stenosing colloid carcinomatous degeneration, which results in an enlargement and distortion of the organ and the transformation of its wall into a fibrous capsule. There is no discussion of the microscopical appearances of any of these specimens, and, hence, there is some question as to whether they were really instances of carcinoma, and not mucoid degeneration or dropsy of the appendix.

Kolaczek,[4] in 1875, reported a case of abscess in the right iliac fossa which opened and the fistula failed to heal. At autopsy a carcinoma of the cæcum was found, which connected with the fistula. The base of the appendix opened at the site of the ulcerated carcinoma, but no mention was made of involvement of the appendix.

This case is here mentioned because of the fact that it has been frequently referred to in the literature as an instance of primary carcinoma of the appendix, when it was very evidently an instance of primary carcinoma of the cæcum.

Leichtenstern,[5] in 1876, briefly mentioned statistically the observation of three carcinomata of the appendix. No mention, however, of the character of the neoplasms was made other than that they were carcinomata.

Bierhoff,[6] in 1880, reported a case of a woman of seventy-eight years who presented at autopsy carcinoma of the uterus and left ovary, as well as of the rectum, with metastatic nodules in the liver and spleen. The appendix was normal for three centimetres of the proximal portion. Three centimetres from the base was situated a carcinomatous nodule the size of a hazel-nut. This nodule produced an obstruction of the lumen such that the distal portion of the appendix was distended into a tense sac containing a grayish mucoid material. This case is here referred to because it, too, has been frequently quoted as an instance of primary carcinoma of the appendix, while in all probability the involvement of the appendix was secondary.

Beger,[7] in 1882, reported a case of considerable interest. The patient, a man of forty-seven years, developed three and one-half years previously a tumor in the right inguinal region, which was incised and about a litre of pus evacuated. The wound, however, never closed. No fæcal gases or intestinal contents appear to have escaped through the fistula. At operation the appendix was found attached by its tip to the base of the fistula, and occupied throughout its entire extent by a carcinomatous process. It was six centimetres in length and about the thickness of the finger. At the base of the appendix was a walnut-sized papillary tumor projecting into the cæcum, which presented the same structure as the rest of the new growth, and had evidently

resulted from extension along the appendix. The patient died thirty-six hours after the operation, and at autopsy metastatic involvement of the retroperitoneal glands was found. Microscopical examination of the new growth showed it to be an adenocarcinoma. The writer believed the carcinoma to have begun at the tip and to have gradually extended to the base of the appendix. The case was of especial interest because of the absence of intestinal symptoms in spite of the existence of the tumor for so long a time.

Maydl,[8] in 1883, mentions one case of carcinoma of the appendix in an article upon intestinal carcinoma. No further mention was made, however, of the gross or microscopical appearances of the tumor.

Draper,[9] in 1884, reported a case of a man of sixty-five years who died with obscure abdominal symptoms which did not allow of a clinical diagnosis. At autopsy the ileum above the ileocæcal valve was distended with liquid fæces. Just at the valve, upon its upper surface, three foreign bodies—two small fragments of bone and a prune-stone—were found lying loosely before the opening into the cæcum. The valve was constricted so as to admit the tip of the index-finger only with difficulty. The mucous membrane of the cæcum and lower part of the ascending colon was thickened and deeply reddened, but not ulcerated. The upper third or head of the appendix was enlarged and dilated to about the size of a large plum. The opening of the appendix into the intestine was also dilated. The thickened wall of this enlarged portion of the appendix presented the characteristic appearances of colloid disease. The free end of the appendix beyond the dilated portion was very slightly enlarged and contained inspissated fæcal matter. The peritoneum and subperitoneal tissues adjacent to the new growth were normal.

In this case there would appear to be a reasonable question as to whether the neoplasm may not have been primary about the ileocæcal valve and to have involved the appendix secondarily.

Lafforgue,[10] in 1893, presented an interesting thesis upon primary tumors of the appendix in which several cases in the literature were discussed and the clinical features of the disease were presented. The writer did not, however, report any additional cases.

Glazebrook,[11] in 1895, reported a case of so-called endothelial sarcoma of the appendix, which, because of the as yet unsettled relationship of these tumors to carcinoma, will be briefly referred to. The patient, a man of fifty-five years, died of cerebral hæmorrhage. At autopsy the appendix was in a normal situation, but

bound down by dense adhesions. It was of normal size for three inches from the proximal end, at which point there was an enlargement the size and shape of a pigeon's egg, situated in the anterior wall of the appendix. The mass was hard and fibrous and resembled a scirrhous tumor. After microscopical examination, the tumor was said to be an endothelial sarcoma, the walls of the appendix being infiltrated by nests of irregular cuboidal or cylindrical cells which were thought to be of endothelial origin. There were no evidences of metastases.

Stimson,[12] in 1896, reported a case of carinoma of the appendix in a woman forty-four years of age, who had had a very severe attack of appendicitis ten years previously. She remained well until two months before operation, when she had a mild attack and recovered. Another mild attack led to an operation and the removal of the appendix, which was four inches in length and one inch in thickness, and presented a degenerated carcinoma, the type of which was not mentioned.

Letulle and Weinberg,[13] in 1897, presented a brief discussion of the subject of obliterating appendicitis, based upon the study of twelve cases, two of which presented primary carcinomata of the appendix. They called especial attention to the development of carcinoma in the fibrous cicatrix of an obliterated appendix and the involvement of all the layers of the appendix by the carcinoma. In one of their cases, both of which occurred in individuals dead of tuberculosis, they were able to demonstrate the starting-point of the carcinoma at the line of fusion where the lumen had been obliterated. In this case the carcinoma had extended from this point through the different layers to the peritoneum. In the second case the terminal extremity of the appendix was the seat of a peculiar adenomatous hypertrophy. The carcinoma had begun in the scar and had involved all the coats of the appendix.

Mossé and Daunic,[14] in 1897, reported a case of primary carcinoma of the appendix, found at autopsy upon a woman fifty years of age, dead of heart disease. There had been, so far as was known, no symptoms of appendicular disease. The appendix pointed downward and inward, was free from adhesions, and was provided with a normal-looking mesentery. It was four centimetres in length and much thicker than normal, having a maximum circumference of forty-five millimetres, cylindrical in shape, about the size of a date and largest at its free extremity. The consistency was firm and the surface smooth. The tumor was entirely confined to the appendix, and was situated for the most part in the mucosa, and in the centre of the growth was the lumen, which was much reduced in size. The mucosa was quite

extensively invaded, the musculature less so, while the peritoneum appeared to be uninvolved. In the vicinity of the lumen vestiges of the glands of Lieberkühn could be made out, some of which had undergone carcinomatous degeneration and seemed to have been the starting-point of the tumor. In portions of the section the tumor presented the appearance of a cylindrical-cell carcinoma, while in other portions it presented more of an alveolar arrangement. The absence of foci in the cæcum, lymphatic glands, and the other viscera led the writer to assume the tumor to be a primary carcinoma of the appendix.

Wright,[15] of 1898, reported an autopsy upon a case of general peritonitis of obscure origin. The autopsy failed to reveal any definite starting-point of the peritonitis. There were some adhesions about the appendix, but no definite evidence of perforation. Upon microscopical examination of the appendix a small primary carcinoma of the head of the organ was found, and just at the junction of the tumor with the wall of the gut was a small perforation which was doubtless the starting-point of the peritonitis. The tumor presented the microscopical appearances of a typical adenocarcinoma.

Monks,[16] in 1899, reported a case of tumor involving the cæcum, in the middle of which there was a slough which seemed to represent the appendix. The tumor proved to be a carcinoma and was undoubtedly primary in the cæcum, with possibly secondary involvement of the appendix. Reference is made to this case because it has been erroneously quoted as an instance of primary carcinoma of the appendix.

Nothnagel,[17] of 1898, briefly mentioned one case of carcinoma of the appendix observed in the Pathological Institute of the general hospital at Vienna. There was, however, no discussion of the characteristics of the case.

Zeman, of Vienna, has also mentioned statistically a case of carcinoma of the appendix, which, however, may be a case already referred to.

Hurdon,[18] in 1900, reported a case of a woman of twenty-four years, who, since the birth of her first child eight years previously, had had considerable pain in the lower abdomen and back, associated latterly with a constant aching in the right lumbar region. The uterus was found to be acutely retroflexed and operation advised. At operation the appendix was found to project downward over the brim of the pelvis and to be involved in dense adhesions. The appendix was removed, and the patient made a good recovery. The appendix, which was ten centimetres in length, was found to be acutely flexed upon itself at about the junction of the middle and distal thirds. The proximal end was

normal, but the distal end beyond the flexion was distended, and contained a soft concretion about the size of a date-stone. Joining the distended extremity to the normal proximal portion was an intermediary portion one and one-half centimetres in length, and of very firm consistence. This on section was found to be a small oval tumor ten by five millimetres, which had produced a marked stenosis of the lumen. Histologically, the tumor proved to be an adenocarcinoma which had invaded all the coats of the appendix. The patient was in excellent health at the time the report was made.

Letulle and Weinberg,[19] in a communication to the Anatomical Society of Paris in 1900, reported two additional cases of primary carcinoma of the appendix complicating chronic appendicitis. In the first case the carcinoma was discovered more or less accidentally in the course of an autopsy upon an individual who had died of tuberculosis. The second case was a child of twelve years of age operated upon by Jalaguier. The patient had had several attacks of appendicitis. The carcinoma, which was of the adenomatous type, was located at a point of the appendix at which a stenosis had resulted from the chronic inflammatory process. The rest of the appendix presented the usual appearance of acute appendicitis. The patient made a good recovery.

Giscard,[20] in 1900, reported a case of a man of thirty-seven years who in March, 1898, had his first attack, which was mild in character. In October of the same year he had a second attack, and after several days developed grave symptoms, which led to an operation. An abscess of the right iliac fossa was found with local peritonitis. The appendix was situated behind and to the inside of the cæcum. The patient made a good recovery. The appendix was about the size of a crayon of chalk, and the lumen was obstructed at about the middle of the organ by what appeared to be a cicatricial thickening. In the distal end of the appendix there was some pus. Histological examination of the appendix showed both a catarrhal and chronic inflammation. At one point the sections also showed a narrowing of the lumen by a neoplasm situated between the mucosa and musculature. This growth occupied about one-half of the circumference of the appendix, and caused a projection towards the lumen as well as towards the periphery. The carcinoma seemed to originate from the deeper layer of the glands, and in its superficial portion presented the characteristics of an adenocarcinoma, while in the deeper portion it resembled an alveolar carcinoma with cylindrical cells.

Rolleston,[21] in 1900, reported a case of a woman twenty-six years of age, operated upon during a fourth attack of appendi-

citis, the previous three attacks having occurred within a period of fifteen months. The appendix was slightly adherent to the posterior wall of the uterus, and on section presented a globular mass a little larger than a marble, and situated near the tip. This mass presented a caseous appearance which suggested tuberculosis. Histologically, it proved to be a spheroidal-cell carcinoma, which in places appeared to extend almost to the peritoneum. The growth was undoubtedly primary in the mucosa. Several months after the operation the patient was reported to be in poor health, and the probability of secondary growths was entertained.

Whipham,[22] in 1901, reported a case of a woman of forty-five years who was admitted to St. George's Hospital with great enlargement of the abdomen and a tumor in the left iliac fossa. Operation was deemed inadvisable, and the patient died four weeks later. At autopsy the abdomen was found to contain a large quantity of serous fluid. The peritoneum over both the parietes and viscera was thickly studded with nodules of new growth. One or two nodules were found in the liver, and the left ovary was transformed into a mass of new growth measuring six by four inches. The lymphatic glands of the neck and anterior mediastinum were also involved. The mucous membrane of the entire alimentary tract was normal, with the exception of a small portion at the base of the appendix, which was occupied by a new growth. The neoplasm proved to be a spheroidal-cell carcinoma. The writer assumed the neoplasm to be primary in the appendix because of the absence of new growths elsewhere in the alimentary tract, and also because the growth was most marked in the mucosa and submucosa, and invaded the muscular coats of the appendix but slightly. The reasons for the assumption that this was a primary carcinoma of the appendix do not appear to be by any means valid, and it is much more probable that the growth originated in the left ovary and metastasized to the appendix.

McBurney,[23] in 1901, reported two cases of primary carcinoma of the appendix. The first was a case of a woman twenty-three years of age, who had had a severe attack of appendicitis two years previously. The symptoms subsided in ten days, and the patient remained well except for a feeling of pain and discomfort in the right iliac fossa on movement or active exercise. Two months before operation the pain became very severe and incapacitated the patient, without rise of temperature or pulse. On examination the patient appeared to be in good health, but the appendix was very sensitive. At operation the appendix was found free from adhesions or disturbance of the peritoneal surface. The organ was four inches in length, much thickened and

enlarged, and presented two strictures, one near the base and one near the tip. Near the tip was a small tumor about the size of a green pea, of dense consistency and white color, which microscopically proved to be a pure carcinoma. There was no evidence of malignant disease elsewhere in the body. The second case was of a man of about thirty years who had given no history of appendicitis. At autopsy the appendix was found to present a rounded tumor near the tip. This tumor was considerably larger than that in the first case, and microscopically was found to be a pure carcinoma resembling the first specimen.

Goffe,[24] in 1901, reported a case of a Jewess of fifteen years, well developed and well nourished, who for more than a year had complained of pain in the region of the appendix after exercise. A clinical diagnosis of chronic appendicitis was made and the organ removed. The appendix was unusually long, thickened, and tortuous, and in the extreme tip was a small white tumor the size of a large pea. Microscopically, the tumor resembled a fibroma, and appeared to have developed in the wall of the appendix and protruded into the lumen. Histologically, it was found to be a carcinoma, which did not, however, invade the muscular coats.

Kelly,[25] in 1901, reported three cases of primary carcinoma and one case of primary endothelioma of the appendix. In the first case the appendix presented the usual character of acute ulcerative appendicitis. At about the junction of the middle and distal thirds was a tumor six millimetres in diameter, occupying chiefly the mucosa and submucosa, and microscopically of the type of carcinoma simplex. The second case was that of an individual twenty-four years of age, with a history of four attacks of appendicitis in the year previous to operation. At operation the appendix was free from adhesions, nine centimetres in length and from five to seven millimetres in diameter. There was no macroscopical evidence of tumor formation; microscopically, however, there was an area near the base of the organ which presented an appearance which the writer considered to be endothelioma. The description of the specimen, together with the uncertain position of this group of tumors, leaves some doubt as to whether this may not have been a case of carcinoma rather than endothelioma. The third case was that of a man of nineteen years who had always been strong and well. Eight days before admission to the hospital he had been taken with severe abdominal pain, which localized itself in the right iliac fossa, where a mass about three inches in diameter could be felt. At operation a collection of pus was found around the head of the cæcum and the base of the appendix. The appendix was five centimetres in length and one

centimetre in diameter. Histologically, the organ presented the lesions of ulcerative appendicitis, in addition to which there was a small growth situated near the base of the appendix. The growth, which was of the type of carcinoma simplex, was located almost entirely within the submucosa, although in a few places there was a slight infiltration of the muscular coats. The fourth case was of a man of sixty-three years, who had always been well until a short time before admission to the hospital, when he began to have attacks of rather severe pain in the right iliac fossa. At operation the appendix was found to be adherent to the surrounding structures, and both the appendix and intestines were studded with numerous whitish nodules suggestive of miliary tubercles. The retroperitoneal glands were also enlarged. The patient died seven days after operation, but no autopsy was obtained. The appendix was two centimetres in length and varied in diameter from one to two centimetres. It presented a constriction at about the middle. Microscopically, the lumen was obliterated, and there was no evidence of a mucous membrane in any part of the organ. The submucosa, muscularis, and peritoneum were infiltrated with nests of carcinoma cells. The meso-appendix was similarly infiltrated. The writer suggests the possibility that the appendix was involved secondarily to carcinoma somewhere else in the body; but the absence of the mucosa and the arrangement of the carcinoma cells inclined him to the view that the growth was primary in the appendix. In view of the fact, however, that there was general peritoneal involvement which was also evident upon the surface of the appendix, together with involvement of the retroperitoneal glands, it would seem very much more probable that the growth in the appendix was secondary to a neoplasm elsewhere in the body.

Harte and Willson,[26] in 1902, have reported two cases of primary carcinoma of the appendix. The first case was of a woman of twenty-four years, who at the age of nineteen had an attack of what appeared to be appendicitis, from which she recovered and remained well for four years, when she had another attack. For several months prior to the operation she had had more or less pain in the region of the appendix. On physical examination there was abnormal sensitiveness in the right iliac fossa and some thickening of the tissues about the appendix. At operation the appendix was found to be free from adhesions and to project upward behind the cæcum. It was fifteen centimetres in length, contained two small concretions, and appeared normal to the naked eye. The lumen was, however, obliterated for almost the entire length of the organ. On physical examination a scirrhous carcinoma five millimetres in diameter was found about

one centimetre from the tip. The carcinoma appeared to have originated from the remains of the glands of the mucosa and to have invaded all the coats of the appendix. The second case was of a man of twenty-five years, who for eight months prior to the operation had had more or less continuous pain in the right iliac fossa. The appendix was found to be bound down behind the cæcum by old adhesions. It contained a concretion about the size of a grape-seed and presented a perforation near the tip. Sections of the appendix about one centimetre from the tip showed a carcinoma taking its origin from the mucosa. All the coats of the appendix were involved in the growth, which was a carcinoma simplex in type. In addition, the appendix presented the gross and microscopical appearance of acute suppurative appendicitis.

Weir,[27] in a discussion of primary carcinoma of the appendix at the meeting of the American Surgical Association in 1902, briefly reported one case which had occurred in his practice. There was, however, no description of the gross or microscopical appearance of the tumor.

Jessup,[28] in 1902, reported a case of a woman of thirty-six years who had had considerable pain in the left inguinal region following an abortion. Operation was undertaken by Dr. Cleveland for disease of the uterine adnexa. A cyst of one ovary was found, and the appendix, which was bound down by adhesions, was removed. The appendix was six centimetres in length, and at the junction of the middle and distal thirds was bent at a right angle, with a constriction at the bend, beyond which was an enlargement. The diameter of this portion was one centimetre while that of the proximal portion was five millimetres. The lumen was obliterated at the bend, and the enlarged portion was occupied by a firm tumor mass, the muscular coat presenting a thin shell. Microscopical examination showed the tumor to be an adenocarcinoma which had infiltrated the mucosa, submucosa, and muscularis. The middle and proximal portions of the organ were free from new growth. There had been no symptoms pointing to disease of the appendix, and the discovery of the carcinoma was accidental.

Of the forty cases here referred to, it would appear that eight were probably not cases of primary carcinoma of the appendix. These eight comprise one case of Merling, one case of Prus, one case of Kolaczek, one case of Bierhoff, one case of Draper, one case of Monks, one case of Whipham, and one case of Kelly. To these eight cases may be added the case reported by Glazebrook as endothelial sarcoma and one reported by Kelly as endothelioma: although it would seem, from the description, that these

may have been instances of primary carcinoma of the appendix. Of the remaining thirty cases there may be some question as to the authenticity of the four cases reported by Rokitansky, the three cases reported by Leichtenstern, the case reported by Maydl, the case reported by Nothnagel, and the case reported by Zeman, because in none of these cases was there a report of a microscopical examination or of positive proof that if a carcinoma existed it was necessarily primary in the appendix.

The remaining twenty cases would, however, appear to be fairly definitely proven to be instances of primary carcinoma of the appendix. The macroscopical findings in these twenty cases are furthermore confirmed by more or less extensive descriptions of the microscopical characters of the neoplasms. These twenty cases comprise one case of Beger, one case of Stimson, one case of Mossé and Daunic, one case of Wright, one case of Hurdon, four cases of Letulle and Weinberg, one case of Giscard, one case of Rolleston, two cases of McBurney, two cases of Harte and Willson, one case of Goffe, two cases of Kelly, one case of Weir, and one case of Jessup.

To these the writer wishes to add the following three cases recently studied by himself.

CASE I. Figs. 1, 2 and 3.—W. D., male, aged eighty-one years, a patient of Dr. Vander Veer. The patient had always been strong and healthy until during the later years of life, when he had shown evidence of both pulmonary and cardiac disease, which were the immediate cause of death. There had never been any symptoms of disease of the appendix. At autopsy, the main lesions were pulmonary tuberculosis with pleural effusion upon the right side, general arterial sclerosis with hypertrophy and dilatation of the heart, chronic interstitial nephritis, and a primary neoplasm of the vermiform appendix. The appendix projected upward behind the cæcum, was free from adhesions, and measured five and one-half centimeters in length. The proximal two centimeters of the organ was of normal appearance and measured six millimeters in diameter. The distal three and one-half centimeters of the appendix was much enlarged and measured three centimeters in diameter. Projecting from the convex surface of the enlarged portion of the appendix at about the middle of the surface, opposite the mesenteric attachment, was a mass of yellowish-green, translucent, gelatinous material. This mass measured two and one-half by two and one-half by two centimeters in its diameters, and presented an irregular contour. On transverse section of the appendix through the middle of the enlarged portion, the lumen of the organ was found to be filled with a gelatinous substance resembling that already mentioned. At about the middle of the convex surface of the enlarged portion of the appendix and opposite the mesenteric attachment was a perforation one centimeter in diameter, through which the gelatinous material in the lumen of the organ was continuous with that already described as attached to the surface. The perforation was definitely circumscribed and the edges were rather firm. There was no evidence of necrosis

of the tissue about the perforation. The gelatinous substance within the appendix presented a striated appearance, the striæ tending to converge towards the perforation. This gelatinous material could be readily detached from the wall of the appendix, and in places presented a decidedly lamellated appearance in addition to the striation already mentioned, and the consistence was about that of calves'-foot jelly. The wall of the appendix, except in the region of the perforation, appeared distinctly thickened, and presented the appearance of an hypertrophy of the musculature. Careful examination of all the other viscera of the body, as well as of the regionary lymphatic glands, failed to reveal any other focus of new growth.

Microscopical examination of sections stained in hæmatoxylin and eosin, as well as van Gieson's stain, did not reveal in any portion of the dilated appendix any evidence of the muscosa. This appeared to be mainly due to an atrophy of the mucosa, resulting from the pressure of the contents of the organ and to a much lesser extent to a destruction of the mucosa by a new growth, which in places could be seen invading both the submucosa and the musculature, but nowhere invading the deeper layers of the musculature or peritoneum, except in the vicinity of the perforation. The new growth presented the typical appearance of an adenocarcinoma, which, however, was evident only in the most recent portions of the growth, and was in no place at all abundant. The new growth was composed of glandular structures which were closely arranged, with but very little stroma. The glands were lined by a high columnar epithelium, the protoplasm of which stained well. In the most recent portions of the growth there was but little evidence of the gelatinous material; but as one traced the glandular structures into the older portions, the columnar epithelial cells became much elongated and the protoplasm stained faintly. The limiting membrane of certain of the cells appeared to have burst, and the contents were extruded into the lumen of the gland. The nuclei of the cells stained less deeply, and began to show slight evidences of fragmentation and disintegration. Gradually the cells became transformed into the gelatinous material, until in older portions of the tumor the individual cells could no longer be distinguished and the nuclei, fragmented and disintegrated, were scattered in an irregular row along the few strands of stroma, while practically the entire gland spaces were occupied by the gelatinous material. Still older portions of the new growth, which to the naked eye appeared to be composed almost entirely of gelatinous material, presented somewhat of a lamellated appearance, the lamellæ being composed of the gelatinous material, while between the lamellæ vestiges of the stroma could be distinguished, associated with which were bits of the fragmented nuclei of the tumor cells. In the oldest portions of the tumor these lamellæ were closely packed together; the result, evidently, of pressure caused by the constant production of the material by the new growth. Between these lamellæ, which corresponded to the much altered gland spaces, the stroma could no longer be distinguished and the fragmented nuclei had entirely disappeared. In the place of the stroma and fragmented nuclei there was a small quantity of rather granular material, which stained deeply with eosin and was probably hyaline in character. The new growth appeared to be fairly well

Fig. 1.—Case I., primary colloid carcinoma of appendix. Showing the projection of the colloid material through a perforation of the organ. (Natural size.)

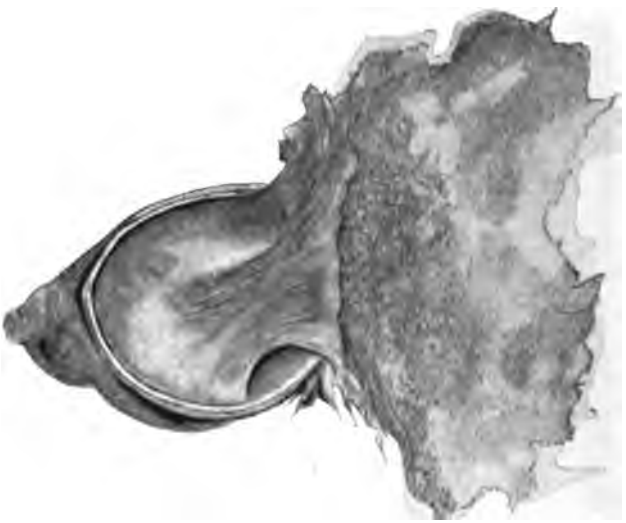

FIG. 2 Case I., primary colloid carcinoma of appendix. Transverse section through the appendix in the region of the perforation. (Natural size.)

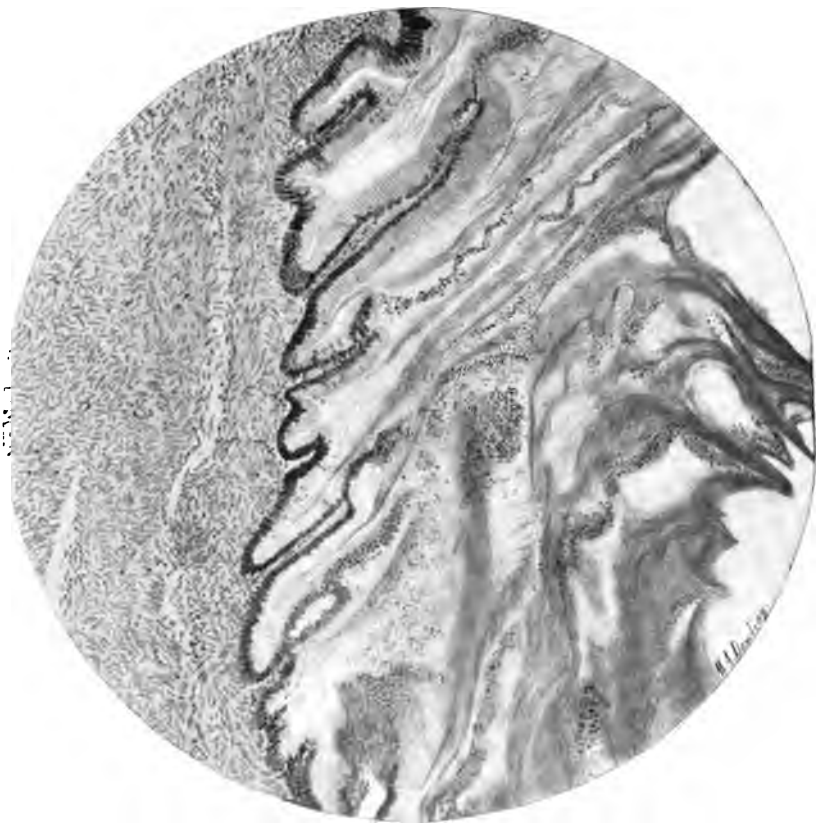

FIG. 3.—Case I, primary colloid carcinoma of appendix.

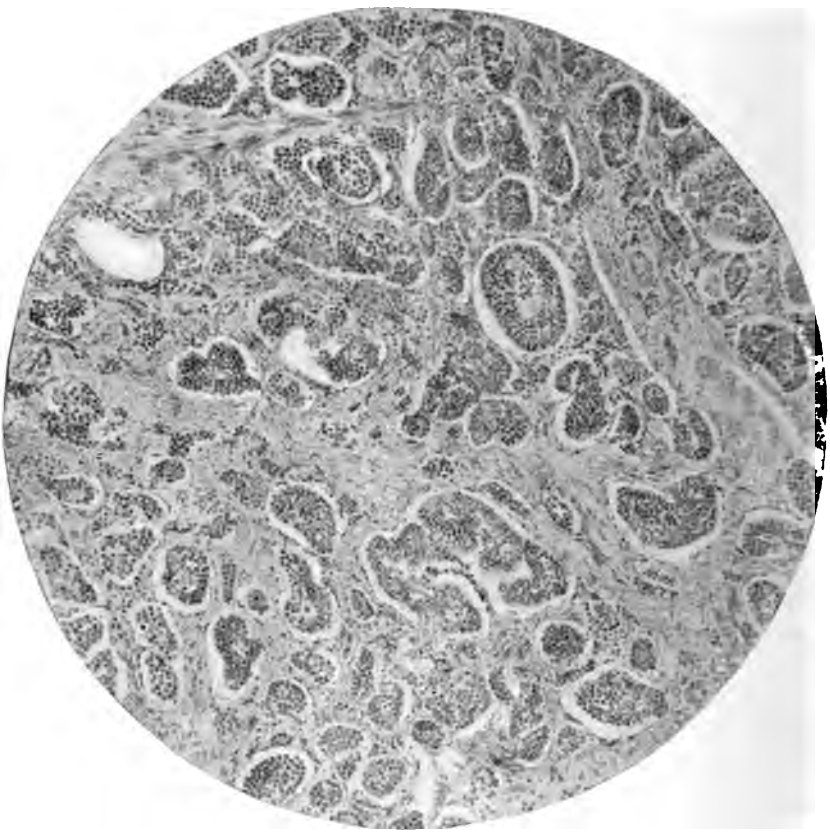

FIG. 4.—Case II., primary carcinoma of an obliterated appendix.

localized in the region of the perforation and did not involve the wall of the appendix at all extensively. Inasmuch, therefore, as there was no evidence of the neoplasm in other parts of the body, the conclusion would seem to be justified that this was a case of primary adenocarcinoma of the appendix belonging to the type which is usually known as colloid carcinoma.

CASE II, Fig. 4 — Mr. L., aged thirty-six years, a patient of Dr. Macdonald. The patient had always enjoyed good health until about eight years previously, when she had an attack of what was called "peritonitis," from which she made a fairly good recovery, and had enjoyed good health until a short time before the operation, when she developed symptoms of pelvic disease. The patient had never manifested any evidence of disease of the appendix. At the operation a cyst of the right ovary was found associated with rather extensive pelvic inflammatory disease, for which bilateral salpingo-oöphorectomy was done. The appendix was free from adhesions and did not present any definite evidence of disease, but was removed in the course of the operation. The patient made an uneventful recovery, and has remained in perfect health ever since, the operation having been done in 1900.

The appendix measured four centimetres in length and ranged from six to nine millimetres in diameter. The mesentery which contained a considerable amount of fat, extended to the tip; the peritoneum was smooth and glistening; the vessels, however, were somewhat congested. Situated about one centimetre from the proximal end of the appendix was a slight enlargement which extended for about eight millimetres. It was in this region that the organ presented the greatest transverse diameter. The consistency of this portion resembled that of the rest of the appendix, which was decidedly firm. The lumen was completely obliterated throughout the entire organ. Transverse section of the base and tip of the appendix showed the lumen to be obliterated by a white reticulated tissue, in the meshes of which collections of fat could be seen. The muscular coat appeared to be normal.

Section through the enlargement already referred to showed the central portion of the organ to be occupied by a more homogeneous tissue, which in the specimen hardened in Orth's fluid presented a slight yellowish tinge. This tissue appeared to extend into the musculature, especially upon the side of the mesenteric attachment. Microsopical examination of sections through the proximal and distal portions of the appendix stained in hæmatoxylin and eosin showed the usual appearance of chronic obliterative appendicitis. The lumen could not be distinguished, and its place, as well as that of the mucosa, was occupied by rather loose connective tissue continuous with the submucosa. No vestiges of the mucosa, glandular structures, or lymphoid apparatus of the appendix could be distinguished. The muscular coats were somewhat thickened and hypertrophied, and the peritoneum was also slightly thickened. In sections through the enlargement of the appendix already mentioned the central portion of the organ was found to be rather densely infiltrated with a new growth, which involved to a lesser extent the musculature and peritoneum. The growth was characterized by numerous large and small alveoli occupied by cells, the outlines of which could be distinguished with difficulty, but which appeared to be for the most part cuboidal or polygonal in shape. These cells contained rather

large round or oval nuclei, most of which stained well in the hæmatoxylin, but some of which were rather vesicular in character. The cells and nuclei appeared to be fairly uniform size, and in most of the alveoli were densely packed together. Some of the alveoli were so small as to present only three or four cells in transverse section. In many of the larger alveoli the process of fixing and hardening the tissue had caused the cells to shrink away *en masse* from the connective-tissue stroma, leaving a clear space. In certain of the alveoli a distinct lumen could be distinguished surrounded by one or two rows of cuboidal cells. Some of the larger alveoli contained a considerable number of red blood-corpuscles which may have found their way there either from trauma at the time of the operation or from invasion of the blood-vessels by the new growth, which in some places was quite evident. The protoplasm of the cells in some of the alveoli presented a small amount of rather diffuse yellowish-brown pigment. The outer or longitudinal layer of muscle appeared to be more extensively involved than the inner or circular layer, and both layers were most extensively involved on the side of the mesenteric attachment. The peritoneum, which was decidedly thickened, also presented numerous alveoli of tumor cells. The tumor was not circumscribed, but appeared to extend rather diffusely through the structures of the appendix, and appeared in places to extend along the blood and lymph channels. In general, the tumor appeared to be epithelial in character, although there were certain portions of it in which an endothelioma of blood-vascular origin was strongly suggested. It is our belief, however, from a careful study of many sections, that the tumor was a carcinoma of a somewhat alveolar type, and was undoubtedly primary in the appendix.

CASE III.—F. C., male, aged nineteen years, a patient of Dr. Macdonald, was admitted to the Albany Hospital, June 9, 1902, complaining of general abdominal pain. The family history was negative, as was patient's past history. He had always been strong and healthy until the onset of the present illness.

The present illness began in January, 1902, with an attack of acute appendicitis, which was associated with the formation of an indurated mass in the right iliac fossa, in which after a short time an abcess formed, which opened externally in the right lower quadrant of the abdomen. The sinus still existed when the patient was admitted to the hospital. The tumor mass on the right side never disappeared, and the discharge from the sinus was fairly continuous. The patient had lost forty pounds in weight. There were no symptoms associated with the fistula which sugguested a communication with the bowel. No diarrhœa, but at times constipation. The patient had had more or less continuous pain in the right iliac fossa for several months prior to admission to the hospital. The fæces had never shown any abnormal appearance.

On admission the patient presented a well-marked tumor mass in the right iliac fossa, the size of a large fist. This mass was tender on palpation, and from the sinus there was a purulent discharge. A clinical diagnosis of tuberculosis of the head of the cæcum was made. The patient was operated upon June 11, 1902.

At operation the mass in the right iliac fossa was found to be composed

of a new growth involving the appendix, cæcum, ileum, the ascending and a portion of the tranverse colon, as well as a loop of the jejunum. All of the involved parts were adherent in a mass. It was found necessary to remove the entire cæcum with the remains of the appendix, and about eleven centimetres of the ileum and ascending, as well as of a portion of the transverse colon, and about eighteen centimetres of the jejunum. The appendix was almost entirely destroyed, and the lumen of the proximal portion opened freely into an abscess cavity the size of a small hen's egg, which contained and was lined by necrotic material, and which communicated with the fistula which opened upon the surface of the abdomen. The appendix and the tissues immediately surrounding it apeared to be the oldest portions of the new growth. There was also extensive involvement of the regional lymphatic glands, which were removed so far as possible. The severed ends of the jejunum were united by a Murphy button with secondary Lembert stutures. The end of the transverse colon was closed, while the end of the ileum was brought out into the wound. The patient did fairly well for about two weeks, when he began to grow weaker and died of inanition.

The specimen removed at operation consisted of the cæcum, the remains of the appendix, about eleven centimetres of the ileum, the ascending and a portion of the transverse colon, and some enlarged lympatic glands in one mass, while in a separate mass were about eighteen centimetres of the jejunum. The distal portion of the appendix was entirely destroyed, while the proximal two centimetres of the organ could still be distinguished. The lumen opened freely into a small cavity two and one-half centimetres in diameter, situated between the ileum and the cæcum. This cavity was located in the mass of new growth, which appeared to correspond to the distal portion of the appendix, and which seemed to be the oldest portion of the tumor. The lumen of the appendix opened freely into the cæcum as well as into the small cavity already described. To this mass of new growth the lower portion of the ileum as well as the ascending and transverse colon were adherent, and were apparently extensively involved. The growth had caused an ulceration through the wall of the ileum as well as of the transverse colon, both of which perforations opened into the cavity already referred to. The new growth was very much more extensive in the peritoneal and muscular coats of the involved bowel than in the mucosa, and it seemed apparent that the adherent intestines had become involved secondarily. The new growth was of rather soft consistence, and on section of a grayish-white color, and appeared to be largely composed of a gelatinous translucent material resembling colloid. In portions of the tumor there was also marked necrosis and softening. The gelatinous material was contained in more or less definite spaces, separated by bands of connective tissue. The lympathic glands were much enlarged and diffusely involved by the new growth, which presented extensive colloid degeneration.

Microscopical examination of sections through the base of the appendix stained in hæmatoxylin and eosin showed a decided thickening of all the coats of the organ, due to a diffuse infiltration by a new growth. The lumen contained a small amount of necrotic material. The lining epithelium had disappeared, but certain of the glands as well as some of the lymphoid

tissue of the mucosa could still be distinguished. The new growth was for the most part of a somewhat glandular type and presented larger and smaller alveoli, which in the more recent portions of the growth were occupied by irregular shaped cells, most of which were cuboidal or polygonal in shape. In some of the alveoli a definite lumen could be distinguished, while in others none could be seen, and the entire alveolus was packed with epithlial cells. In some of the places there was a slight resemblance between the glands of the mucosa and the more recent portions of the growth. In certain portions the tumor was composed simply of narrow columns of epithelial cells, suggesting the appearance seen in carcinoma simplex. In older portions of the growth the alveoli were much larger, and the epithelial cells were grouped along the periphery of the alveolus, while the central portion was occupied by a homogeneous substance which stained very faintly with hæmatoxylin, and which presented a marked reticulated appearance, and in which an occasional degenerated epithelial cell or nucleus could be seen. In still older portions of the growth several alveoli had apparently fused, and the intervening stroma as well as the fixed tissue in general had largely disappeared. Most of the epithelial cells had degenerated, and the alveoli were filled with coloid material, scattered through which were occasional more or less degenerated epithelial cells and free nuclei. The colloid material first appeared as small refractile globules in the protoplasm of the tumor cells. These globules enlarged and became fused, as a result of which practically the entire cell came to be occupied by the colloid material, which stained very faintly with hæmatoxylin. The nucleus of the cell, either entirely disappeared or was pushed off to one side of the cell, presenting the signet-ring appearance. The cell membrane in many instances appeared to remain intact, but sooner or later ruptured, and the colloid material became fused with that resulting from the degeneration of neighboring cells. In the oldest portions of the tumor practically all the cells had undergone the colloid degeneration, and the alveoli were occupied simply by the colloid material without, in many instances, a single distinguishable epithelial cell or nucleus. These alveoli showed a marked tendency to fuse, thus giving rise to extensive areas of colloid material. The colloid substances presented a markedly reticulated appearance, part of which seemed to be due to the preservation of more or less of the cell membrane as well as some of the intervening stroma. There were, however, in the oldest portions of the tumor but comparatively little stroma and very few blood-vessels. The lymphatic glands were diffusely infiltrated with the new growth, and only a small zone of the lymphoid tissue remained immediately beneath the capsule. The colloid degeneration was even more marked in the lymphatic glands than in the tumor itself, and practically all of the new growth appeared to have undergone this degeneration.

From a careful, clinical, anatomical, as well as pathological investigation of this case, we feel justified in assuming that it was a primary colloid carcinoma of the appendix of an adenomatous type, with extensive secondary involvement of the neighboring portions of the intestines, as well as the regional lymphatic glands.

The early view was that carcinoma of the appendix was not primary, but resulted from extension from some neighboring organ. On the contrary, recent investigation has shown that primary carcinoma of the appendix is of more frequent occurrence than is ordinarily supposed, while secondary tumors of the appendix are of rare occurrence, even though the cæcum may be extensively involved.

Regarding the etiology of carcinoma of the appendix comparatively little is known, although recent studies have shown that in some instances, at least, it is one of the sequelæ of chronic inflammation of that organ. Theoretically, the appendix should frequently be the site of carcinoma, because certain factors which are usually supposed to bear an important causal relationship to the development of neoplasms are in evidence in this organ. In the first place, carcinoma of the gastro-intestinal tract tends to originate at those portions which are narrow or constricted, which is one of the characteristics of the appendix. Secondly, fœtal remains as well as atrophying organs appear to be more prone to the development of carcinoma, and such a condition is supplied by the appendix. Thirdly, mechanical irritation, which is such an important factor in the development of certain neoplasms, exists extremely frequently in the appendix, and usually results from the action of enteroliths, dried fæcal matter, and occasionally foreign bodies. When one considers the great frequency with which gall-stones are followed by the development of carcinoma of the gall-bladder or bile passages, it seems extremely remarkable that such a condition is not more frequently observed in the appendix. Fourthly, chronic inflammation, which in so many instances is followed by the development of neoplasms, occurs almost as frequently in the appendix as in any organ of the body. The studies of Letulle and Weinberg, Harte and Willson, and others have shown that primary carcinoma of the appendix does occasionally develop in an organ the subject of chronic inflammation, usually of the obliterative type. Of such a character is Case II reported by the writer, in which a typical carcinoma had developed in a completely obliterated appendix. It may furthermore be urged that if more careful routine examination were made of appendices removed at operation, primary carcinoma would be observed more frequently, for in many of the cases reported during the past few years the new growth has been an accidental find, the presence of which was never suspected before operation, or even, in some instances, after macroscopical examination of the organ. It is only comparatively recently that many surgeons have made a practice of having all appendices removed at operation examined by a path-

ologist, and this certainly accounts for the increased number of neoplasms of this type observed of late. Sections should be studied not merely from one or two portions of the organ, but from several portions, and especially in those appendices which show evidences of chronic inflammation.

A striking feature of many of the cases of primary carcinoma of the appendix reported is the devolpment of the disease in comparatively early life. Of the twenty-three cases in which the proof seems conclusive that the new growth was primary in the appendix, the age of the patients is stated in seventeen. Nine of these seventeen, or fifty-three per cent. of the patients, were under thirty years of age, while four, or twenty-four per cent., were under twenty years of age. The youngest case reported was that of a child of twelve years. The early age at which such a large percentage of the cases occurred may be assumed to bear a definite relationship to the age at which appendicitis is most frequent. For, as is well known, the great majority of cases of appendicitis occur in individuals under thirty years of age, and an especially large percentage of the cases are under twenty.

Carcinoma of the appendix may belong to any of the ordinary types of that neoplasm, although the colloid type appears to occur more frequently than any other. In Cases I and II reported by the writer the neoplasms were of that variety. In a considerable number of the cases reported the new growth was confined to the appendix, and did not present any evidence of either extension or metastasis. This was very likely due to the fact that most of the tumors were removed in a comparatively early stage. The tumor may attain considerable size and may ulcerate through the wall of the appendix, and thus give rise to a local or general peritonitis. It may also give rise to a focus of suppuration, which may present the usual characters of an appendicular abscess. By local extension neighboring portions of the intestines or other viscera may become involved, and the pathological picture will depend largely upon the extent of this involvement.

In the great majority of cases the diagnosis of carcinoma of the appendix is impossible. Writers have even gone so far as to state that it is practically *always* impossible. In some of the cases there are no symptoms whatever pointing to the appendix, and the tumor is an accidental find either at operation or autopsy, as instanced by Cases I and II reported by the writer. When symptoms are present, they are usually those of appendicitis of the chronic relapsing type. In some instances the new growth may apparently cause an acute attack of appendicitis, and a perforation may result, usually at the site of the tumor. Pain

is perhaps the one symptom present in the majority of the cases. This is usually referred to the right iliac fossa and may be of very varied character. When present, it differs in no way from that associated with chronic appendicitis, and hence the diagnosis is usually of that condition. The pain is usually due to the mechanical action exercised by the tumor. In the later stages a well-defined tumor mass may present in the right iliac fossa which strongly resembles an appendicular abscess. When it attains considerable size, the tumor often shows a tendency to be associated with the formation of an abscess, which may open externally, and a discharging sinus is formed which shows no disposition to heal. From such a sinus gas and fæcal contents may be discharged, though this is very exceptional. The existence of such a condition with normal defecation might speak in favor of a neoplasm of the appendix. Diarrhœa and constipation, or both, may also occur in the later stages, but they are usually due to the extension of the tumor into the neighboring intestines, and are in no sense the result of the new growth in the appendix itself.

The treatment of the condition is exclusively operative, and since the association of the new growth with the inflammatory process in that organ has come to be so well recognized, there is an added reason for the extirpation of those appendices which present evidences of either acute or chronic inflammation.

From a careful study of the subject, the following conclusions may be drawn:

1. Primary carcinoma of the appendix is not of such rare occurrence as has been hitherto supposed.

2. Every appendix removed at operation or autopsy, if it presents any evidence whatever of disease, should be examined most carefully, and sections should be made from several portions of the organ for microscopical study.

3. The relationship of primary carcinoma of the appendix to chronic appendicitis, especially of the obliterative type, seems to be fairly definitely established.

4. Primary carcinoma of the appendix shows a tendency to develop at a comparatively early period of life.

5. Primary carcinoma of the appendix does not show a marked tendency either to extension or to metastasis.

6. The symptoms of primary carcinoma of the appendix are usually the symptoms of appendicitis of the chronic type.

7. The diagnosis of primary carcinoma of the appendix is in the great majority of cases impossible.

8. The treatment of the condition should always be operative.

[The writer wishes to acknowledge his indebtedness to Dr. W. W. Sanford and Miss M. A. Dowling for the drawings published in connection with this article.]

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DISCUSSION.

Dr. WILLIS G. MACDONALD, of Albany: This very important paper is worthy of some discussion, and as an Albanian I am proud that it represents a class of work in this city in which the surgeon and pathologist are associated. Reference has been made to the extreme rarity of carcinoma of the appendix, but I do not believe it is anything like so infrequent as the literature appears to indicate. The systematic and thorough examination of every appendix removed by operation will surely lead to the discovery of many more cases. Of course, in the case presenting profound symptoms surgery can only hold out a forlorn hope. In a case coming under my care for the relief of a pelvic condition associated with fibroma, and having a previous history pointing to appendicitis, I determined to look for the appendix after having attended to the condition of the pelvis. The latter appeared to be the seat of an ordinary chronic inflammation, but it was removed, and was found to be the seat of carcinoma. The woman has recently written to me reporting that she is in excellent health. This is a most gratifying result, and shows the importance of seizing the opportunity, when opening the abdominal cavity for other causes, to look for disease of the appendix. Co-operation between the surgeon and the pathologist is most praiseworthy, although, I must confess, occasionally it is most disappointing and discouraging to the surgeon to receive a report from the laboratory stating that the surgeon has removed a normal appendix.

XXXV. CANCER OF THE CERVIX. TREATMENT BY THE X-RAY. REPORT OF CASES

By THOMAS P. SCULLY, M. D.

ROME

Surgeons, as a rule, are of the opinion that cancer of the cervix uteri, as it is usually met with, has advanced beyond the stage when any operation can be more than palliative; for the above reason any procedure that affords greater promise of cure is eagerly seized upon by both patient and surgeon. Various expedients have been from time to time recommended and used, but nearly all have fallen short of the promised relief and gradually been discarded.

When we speak of cancer of the cervix uteri, the whole question of cancer of the uterus and its appendages naturally is thought of, for it seems to be pretty generally conceded that in the great majority of cases of cancer of the uterus the primary point of attack is on the os or cervix. Of all the conditions which the gynecological surgeon is called upon to treat or operate upon, there is none quite so terrible, or more painful in all its manifestations as cancer of the uterus and its appendages; furthermore, there is no condition where in the major proportion of cases we are so helpless or powerless to bring about a radical cure. In cases where the condition is recognized in time and radical operation is performed, a cure may be brought about, but nothing short of a hysterectomy is of any use where a diagnosis of cancer of the cervix is made by aid of the microscope. I have performed hysterectomy in quite a number of cases for cancer of the cervix in the early stages with fairly good results. There is no question, if statistics count for anything, that cancer of the uterus is on the increase, consequently, it behoves all physicians, particularly the family physician, to be ever on the lookout for the earliest indications of symptoms of cancer of the cervix and inform his patient of the true condition and its gravity and the treatment appropriate, and under no circumstances should temporizing expedients be resorted to or advised, once the condition is recognized, less the surgeon be called in the case when the disease has progressed to that stage when it may be too late to do more than a palliative operation. Like all who are following along special lines, I have had an opportunity to see many of those extremely painful conditions of this dreadful disease and have used the various expedients at our command many

times as I have stated, in vain, and have longed for some other means to help these unfortunate individuals.

During the last year several men have been working along the line of investigation with use of the X-ray in such places within the body as the rectum and vagina, and while unaware of each other's endeavors all have arrived at practically the same conclusion as to the manner in which the rays could be used successfully.

E. W. Caldwell, B. S., New York city, director of Edward N. Gibbs X-Ray Laboratory, University and Bellevue Medical College, describes in an article published in *New York Journal*, November 22nd, 1902, a specially constructed tube. It is a very unique contrivance and certainly has many excellent features to recommend it. Prof. Pennington of Chicago has devised a shield of metal which is clasped around an X-ray tube; this shield has a cylindrical prolongation which can be used as a speculum or to which a speculum can be attached and through which the rays are concentrated on growths in rectum or vagina.

CASE No. 1. Several months ago, Mrs. A. W., aged forty-six, mother of seven children, consulted me for uterine trouble, which proved, on examination, to be an advanced case of cancer of the cervix of the variety known as the cauliflower growth, and gave a history which did not differ from that usually given in similar cases. She would not hear to an operation, consequently, some other measure that could be used if only for temporary relief was what she desired. It occurred to me to apply the X-ray, and when I explained to her what I proposed to do, mentioning the use of the X-ray in connection with such conditions in other localities, she concluded to have it tried.

I blackened my X-ray tube, with the exception of a small space in front where by rubber bands a Ferguson glass speculum was attached, and by inserting the speculum in the vagina consentrated the X-rays on the growth which protruded from the external os. The treatment was given once in two days, lasting each time from fifteen to twenty-five minutes. After three treatments, a change could be noticed in the discharge, which had been present for some time and which was bloody and very offensive. It became changed to a light yellow and was no longer offensive in odor. At the end of four weeks the mass, which had completely filled the vault of the vagina, was reduced fully one-half and the patient, who had been greatly depressed in spirits and suffered continually with pains in the back which would shoot down the limbs, became very cheerful and hopeful and comparatively free from pain. The treatment was continued every two days for three months, at which time all appearance of growth had disappeared, although there still continued a discharge from the uterus which was not offensive. She had gained in flesh and strength and was feeling in excellent spirits. At this time the patient's daughter, who lived in an adjoining state, met with a serious accident and the mother went to take care of her; hence the treatments were discontinued. She reports at intervals, is feeling splendid,

as she calls it, and believes she is cured. I would like to have continued the treatment for some time longer.

CASE No. 2. While treating case No. 1, shortly after the fourth week of treatment, I was so convinced of the benefit derived from this method that I tried the same on a Mrs. J. B. M., aged forty-three years, mother of five children, whom I had seen a short time previously and who had returned from a hospital in a neighboring city where she had gone for an operation for cancer of the womb, but was informed that nothing could be done for her. I offered her the opportunity of the X-ray treatment, which she readily accepted. Her condition, on examination, was as follows: The whole upper portion of vagina and what remained of the cervix was one big rodent ulcer, from which a very offensive discharge was continually exuding. It had evidently involved the pelvic connective tissue, as the pains in lower portion of the abdomen and bladder were at times very pronounced. The rectum did not seem to be involved, and it appeared as if the involvement extended anteriorly toward the urethra, as at this time she was experiencing pain during urination. Treatment was commenced with X-ray, but there was not such rapid improvement in this case as in case No. 1. In fact, it was not until she had had the rays applied six times that any apparent change took place, and this change was noticed in the freedom from pain in urinating. From this time on the improvement was more pronounced, and the offensive discharge became less and less, and as the healing proceeded it was observed that the cervix was nearly obliterated, only a small portion of what appeared to be a remnant of the posterior lip remaining in sight. In the healing process the cicatrix that formed resembled very much the cicatrix of a burned surface and, of course, seemed very uneven. I have this case still under treatment, as a small part of the anterior portion of the vault of the vagina still remains unhealed. The patient has gained in flesh and is free from pain and gives every indication of being ultimately cured.

CASE No. 3. Mrs. B. D., aged forty-seven, mother of three children; married at the age of twenty-eight; first child born at thirty, forceps delivery, cervix was badly torn at that time. Never had any surgical operation until three years ago, when she was treated for endometritis and curetted, with very little benefit. During the last year the discharge became more offensive, and at times bloody. Did not again consult her physician until two months ago, when she was informed that she had a malignant growth of the womb, and was referred to me by her family physician for the purpose of having an operation, since it was thought possible that removing the womb might save her life. On examination, a growth was found involving all the cervix, the cervix being very large, filling all the vault of the vagina, which of itself was very roomy. The portion of the growth in vicinity of the os had sloughed away, leaving numerous deep cavities from which an offensive discharge came. The tissue broke down very easily by the finger coming in contact with it and bled very freely. The body of the uterus was apparently involved, and the chances for recovery from an hysterectomy did not look encouraging; therefore, I suggested the X-ray treatment, which was accepted. She has had twelve treatments, and the external appearance of the ulcer shows a

diminution in size and cicatrization is taking place. She is under treatment at present and certainly gives every indication of having a good result. I have two other cases under treatment with about the same history as case No. 3. Have given only a few treatments, therefore, cannot report on results at present.

The question naturally arises as to whether I consider the results in such a limited number of cases of sufficient importance to warrant me in coming to any conclusion as to the advisability of recommending this treatment in all cases of cancer of the cervix, and also as to whether I am justified in recommending this treatment in preference to a radical operation for the removal of the deceased uterus and its appendages. I will state that it is my opinion at the present time that the early stages of cancer of the cervix uteri, nothing short of a complete removal of the uterus should be attempted, but in the later stages of cancer of the cervix where the vaginal vault is involved and also the body of uterus and it is highly improbable that more than temporary benefit can be given the patient by surgical means; therefore, it would certainly seem that the X-ray treatment should be resorted to and judging from the limited experience I have had in its use, I feel justified in saying there should be no hesitancy in using or recommending its use in such cases.

XXXVI. A CONSIDERATION OF THE PATHOLOGY AND TREATMENT OF CANCER.

By LEONARD WEBER, M. D.

NEW YORK

At a meeting in April, 1902, of the American Medical Association of the Greater City of New York, a discussion was had on this subject which induced me to look up the history of my own cases, and inquire at the same time into the views held by some of the best authorities of the present day, in regard to the nature of cancer.

The supposition of a parasitic origin of cancer has been agitating the medical and lay minds considerably of late, but no specific cancer-parasite has yet been found. No doubt, it is the analogy of the invasion of the system from a primary focus in cancer with that of the tumors formerly known as infectious tumors, particularly of tuberculosis, by which the belief in the parasitic theory has been brought about. However, as F. Marchand, in *Deutsch. Med. Woch.* No. 40, 1902, p. 722 says: "Though

there is a similarity in many cases of rapid eruption of cancer in innumerable foci with the course of an infectious process, yet it has been often enough demonstrated, that the metastases of all genuine infections are caused by the isolated parasites, which excite the tissues of any suitable locality to identical changes by their specific actions, and all infectious processes belong essentially to the department of inflammatory and consequently, reactive conditions. In malignant growths, on the contrary, it can be demonstrated with absolute certainty that the metastases originate solely through the progressive multiplication of transplanted tumor-cells, which have no infectious action upon the tissue elements and do not cause a transformation of normal epithelium into cancer cells. This can be very clearly seen in the lymph glands, the brain, etc., where the tissue-elements are sharply separated from the tumor-cells, so that the characteristic point for infection by germs, that is, that the specific infectious germs will produce a specific and always the same morbid process where so ever they get to and find suitable soil, is utterly wanting in the formation of the metastatic tumors of cancer. The tumor-cell itself is a living and reproductive organism, living upon and increasing in the substance of the body. It may be compared in its action and progress to a parasite, but is not identical with it."

There are also certain pathological facts which speak against the parasitic nature of cancer and other malignant growths, particularly the numerous cases of carcinoma out of congenital inclusions or aberrant groups of cells; it would be unreasonable to presume that these epithelia contained parasites already, when segregated. Marchand, Hansemann, Ribbert, Orth and other well-known pathologists make the positive statement that the great majority of cancer parasites which are believed to have been seen of late, are nothing but well known products of degeneration of protoplasm, or vacuoles and impurities within the cell body, and are not deserving any further consideration. A sole exception, perhaps, would be certain corpuscles which have lately been seen and demonstrated by a number of careful observers, such as Plimmer, Borrel, and Leyden, in a series of cancers, particularly of the mamma, and pronounced parasitic by some and not parasitic by others. Some of these specimens did look like parasites, but careful tests and examinations made in the pathological institute in Leipzig by Marchand and Noesske appear to have yielded the results that such corpuscles were not organisms but inclusions of a different kind within the cells, so-called vacuoles, produced by cellular secretion and containing a varying amount of coagulated substance. These investigators

believe that the growth of malignant tumors out of proliferating elements of the adult organism, point to a particular disposition, inclination, and that external causes in the way of acute trauma or chronic irritation by ulceration, etc., serve as irritants only, in-so-far as they excite cellular productiveness, as we see it particularly in advanced life.

Heredity plays a great rôle here, understood as hereditary or acquired morbid disposition of certain tissues. There are many things which Marchand believes, are apt to show that the reappearance of epithelial proliferation in advancing age is closely connected with a state of degeneration,—not of a return to the embryonal state, but a change from the normal condition. In the course of such degeneration or change with age, the cells are apt to escape from the regulating influence of the nervous system and acquire a greater amount of independence of their own. Ribbert thinks that the cells have got to be separated first from their normal surroundings, before they can be stimulated to abnormal proliferation, but this is not absolutely essential, because we can demonstrate the overgrowth, degeneration and productiveness of cells within such glanular acini as are still perfectly well preserved, and this has been done in the mamma, the pancreas and prostatic gland.

To be sure, it is difficult to answer the question what cause may excite again the cells in later life to renewed and increased proliferation, but every attempt at an answer is mere speculation. Hansemann believes that the cause of the malignity lies in the return of the cell to a less differentiated state—anaplasie as he calls it,—histologically perceptible by the appearance of abnormal asymmetrical splitting of its nucleus, the descendants of these unequally divided cells departing further and further from their normally differentiated ancestors. That such pathological anaplastic differentiations of the normal tissue cells are seen in malignant growths, there is no doubt, because we see often enough a complete transformation of the cells of a part when a carcinoma develops in it. But this is by no means always so, and quite often the tumor cells come pretty near the original epidermoid cells in form and arrangement, nor is the degree of malignity dependent upon the more or less pronounced anaplasie of the cells. Very small cancerous tumors of the stomach and intestines, presenting an exquisite picture of glandular construction, may nevertheless produce large metastatic tumors of the same kind. Ribbert's observation of the segregation of groups of epidermal cells from surrounding normal tissue by inflammatory processes, is also of importance in considering the cause of malignant proliferation. There are many

cases of tumor formations which can be traced back to segregated parts of organs and in some of them the separation has been brought about by inflammatory proliferation of connecting tissue, as, for instance, in cutaneous cicatrices. But the process of such separations can only be proved in very few cases of new growth, while in many cases of development of cancer, of the skin for instance, a progressive proliferation of the cells of the epidermis and the glands and the hair follicles can with certainty be observed to take place at various points at one and the same time.

If external causes then, can do no more than excite proliferation of cells, there must necessarily occur abnormal changes in the substance of the cell by which its physiological structure is changed, and such products of cellular actions must have not only a stimulating but also a toxic effect. Without a supposition of such toxic effect, it would be difficult to understand the peculiar destructiveness and even inflammatory properties of the cancer-cells in their invasion of the tissues; we need only think here of pleuritis and peritonitis carcinomatosa. It is believed by Marchand and others that the cells acquire such virulence only after being deprived of certain influences, probably by the nervous system, which are the regulators of the normal cellular functions. These physiological influences may be lost by hereditary or acquired weakness or disturbance of the finest nervous adjustments. It must not be forgotten, however, that many cells functionate very well without such regulations.

We see then, that these well-known investigators are opposed to the parasitic origin of malignant tumors. With the acceptance of Marchand's theory of the toxic properties of the cancer cell, the diminished resistance of the tissues against the luxuriant growth of cancer-elements would assume a less mechanical aspect, while on the other hand, the effectful resistance of the normal organism would consist to a large part in that, that the toxic properties of the proliferating cells can be and are neutralized by the healthy tissue-changes in a sound body.

Now, it is certainly very remarkable that many of the best pathologists of the present day neither believe nor take any hand in the bacteriological investigations of cancer, yet this sort of investigation is the signature of the present times and many experienced clinicians believe in the parasitic nature of the malignant tumors. There is, however, no way of getting round the inherited or acquired disposition of the individual, no matter whether the malignant productiveness of the cells is excited by invading parasites or by a pathological chemistry of the cells alone. Be the one or the other true, it does not signify to the

surgeon for the present and the next and probably remote future, for it will always be his business to extirpate the tumor as soon and as radically as possible. With the proof, however, of the parasitic nature of the disease, the physician would be justified to hope for the early discovery of a reliable healing serum or antitoxin by which the parasite or its toxins, or both, could be destroyed.

It goes without saying that, even with early diagnosis and early and radical operation, the prognosis as to recurrence sooner or later, is still bad and quite uncertain. We have nothing definite by which we can be guided in giving a prognosis, *bona* or *infausta*, in the average case, and I have found it a good rule in practice to be exceedingly cautious in making a definite statement with regard to it to the patient or his friends. There is not time enough to prove this by detailed histories of the many cases which have come under my care in a long practice. Suffice it to say that, in the cases of carcinoma mammæ, many a hopeful case was seen to relapse early and end fatally, in spite of the best surgical procedures, while others again, presenting rather bad local and general features, did well and remained well.

With epithelioma of the lip or face or nose the best results were invariably obtained by early and liberal excisions, much less so by escharotics such as arsenic and chloride of zinc, the use of which I have given up of late and advised against in consultation with professional associates.

Comparatively good results I have seen in about six cases of malignant tumor of the transverse and descending colon which were cut out as soon as the consent of the patient could be obtained. The worst results were noted by me in about eighteen cases of cancer of the stomach, none of which lived longer than eighteen months, counting from the time that the diagnosis could be made, with or without operation.

Though I have not observed a cure of local relapse by the X-ray treatment, in the few cases of mammary carcinoma where I advised the application of the same, I am ready to state that pain and distress have been relieved by the actinic rays, some of the smaller tumors around the cicatrices on the field of operation diminished in size, even disappeared, and the patient's general condition improved.

There is no doubt in my mind that your thoughts did or will correspond to mine when I say in conclusion, that all operable carcinomata ought to be removed by the knife as early and radically as possible. By pretty rapid and dexterous operating and the avoidance of all unnecessary bruising of and pressing into

the affected parts, by which cancer-cells may be driven and dispersed into neighboring tissues, the prospects of smooth healing and non-occurrence of relapses are greatly improved, according to my experience. So soon as the surgeon can discharge the patient he ought to return to the care of his physician for such constitutional and other local,—particularly X-ray after-treatment—as may be indicated by the nature of the case, in order to prevent or postpone relapses as much as may be. In many cases much can be done, I believe, by diet, hydrotherapeutics, frequent exposure of the body to sun or electric light, the tonics of arsenic, iron and chinin towards increasing constitutional resistance against the carcinomatous toxins and the making of a soil unsuited to the proliferation of degenerated cells, or, if, you please, to the growth of cancer parasites.

XXXVII. PLASMODIOPHORA BRASSICÆ (WORONIN.)

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This organism discovered by Woronin in 1876 is the recognized cause of a disease of many of the edible *Cruciferæ*, notably all kinds of cabbage, cauliflower, turnip, radish, kohlrabi, and, according to Tubeuf, has also been found in *Iberis umbellata, Capsella bursa-pastoris, Mathiola incana.* The infection of the vegetables above named by this organism is followed by the development upon the roots of tumor-like outgrowths and swellings of varying size and bizarre form. (Fig. 1.) The foliage of the infected plant is usually stunted and as the disease progresses shows such evident wilting that the infected plants can be readily detected by this means alone. The disease has a very extensive distribution and is of considerable economical importance. It is known in this country as "clubroot," "clumproot" and "clubbing." In England it is usually spoken of as "finger-and-toe disease" or "anbury;" in Russia, where the organism was discovered, "Kapoustnaja Kila;" in Germany as "Kohlhernie," "Kohlkropf" or "Vingerzichte" and in Belgium and France as "maladie digitoire." The distribution of the parasite seems to be as extensive as that of the plants which it infects. The disease produced such ravages in Russia that in 1872 the Royal Association of Gardeners offered a prize for the discovery of the cause and means of prevention. The disease was first

noted in Scotland in 1789. According to Eycleshymer, writing in 1891, the stronghold of the disease in the United States is in New England and the Middle States, especially in Connecticut, Rhode Island, Massachusetts, New Jersey and Delaware and the south-eastern portions of New York and Pennsylvania. It had, at that time, however, extended to Maryland and Virginia and the Carolinas. It had also been noted in Missouri, Wisconsin, Iowa and Michigan. This author has also called attention to the fact that the disease occasionally develops in newly broken ground where no crop has yet been grown and believes that this indicates that certain soils harbor the organism as a saprophyte. The disease is most prevalent in unusually wet seasons and is almost endemic on land which is poorly drained and damp. It seldom occurs where the loam is sandy or calcareous. The same author calls attention to the fact that in the neighborhood of limestone outcroppings the vegetables are comparatively free from attack and states that on Long Island the gardeners raise their cabbages year after year on the old shell heaps without any trace of the disease. The disease is known to occur in fields which have been covered with compost where the adjoining fields which had not been fertilized were free from attack.

The organism which produces these outgrowths and tumor formations presents many points of interest. Woronin, who gave it the name of *Plasmodiophora Brassicæ,* classified it among the *Myxomycetes,* an order of uncertain position, but generally viewed as lying midway between the animal and plant kingdoms.

DeBary,[1] who considers this order to belong more properly to the animal than to the plant kingdom, has suggested the name *Mycetozoa,* which, according to more recent classifications, is preferable. Certain groups of these organisms present characteristics which identify them with the lowest forms of plants, while others present characteristics which unmistakably indicate that they are of protozoan nature. To this last group undoubtedly belongs *Plasmodiophora Brassicæ.* Doflein[2] so classifies the organism and in fact, places the order *Mycetozoa* under the *Rhizopoda.* The lower forms of this order present many points of similarity to the *Amoebæ* and the *Heliozoa,* but differ distinctly from these organisms in their developmental cycle. In fact, very little is known about the entire class and without doubt many points in the cycle still remain to be determined. Doflein divides the order into two sub-orders, *Protomyxidea* and *Mycetozoidea.*

The organism we are considering is placed by this author in the first sub-order. The developmental cycle of these organisms

differs according to their position in the scale. The higher, those which most closely approximate the fungi, present more or less complicated cycles, but in none has a sexual phase been determined. So little is known about them, however, that it is highly probable that we are but imperfectly acquainted with the life cycle of even the lowest forms.

The simplest form of the *Mycetozoa* is found in a minute mass of protoplasm, the so-called swarm spore, which develops into myxamœbæ. In this phase the myxamœbæ may change their form, protrude delicate flagella and are then known as myxoflagellates. The flagellate and amœboid forms are practically interchangeable. As an amœba the organism contains a more or less well-defined nucleus and occasionally a contractile vacuole. It may divide and in this way increase in number; it may become encysted as a single amœba, or certain of the organisms may increase in size until cysts of considerable dimensions are formed in which there develop a number of spores. In all probability this stage is preceded by some form of copulation as yet undetermined. The spores in this case, after liberation from the cyst probably develop within them swarmers, which, on escaping, take on the form of myxamœbæ or flagellates. Fig. 2, adapted from Doflein illustrates what is known of the cycle of the *Protomyxidea*, the sub-order to which *Plasmodiophora* belongs. Question marks inserted in this cycle indicate that possible intermediary stages are as yet undetermined.

The organism *Plasmodiophora* as described by Woronin was first noted in the spore form as minute, spherical, yellowish-green bodies of uniform size, filling the enlarged cells of the infected plant (Fig. 3). These cells were usually arranged in groups. Besides the spores, Woronin recognized the presence in certain cells of large masses of protoplasm more or less filling out the sap spaces of the cell. These he viewed as the plasmodial form of the organism.

As to the ectogenous phase of the parasite, Woronin noted that the spores presented a definite delimiting capsule within which granular protoplasm could be detected. The mass of protoplasm within the cyst later developed into a definite body or swarmer which, on the bursting of the cyst, was liberated and thereupon took on the form of an amœba, showing more or less active amœboid motion, or protruding delicate flagella and moving about the field in the form of a flagellate. Fig. 3 after Woronin, sufficiently illustrates this phase of the organism. Woronin determined that the organism observed was the cause of the tumor-like outgrowths in which they were found, by infecting ground with the dried roots of infected plants. By

so doing he established beyond question the relation of the organisms to the disease.

Upon the disintegration of the roots the spores of the organism remain in the ground until the following season, when, with the advent of warm weather the swarm spores are liberated from the spore cysts and infect the growing plants. The exact method by which the amœboid or flagellate form of the organism enters the uninfected plant has never been definitely determined, but it is generally supposed to be by means of the root hairs. Infection may occur, according to Woronin, at any period during the life of the plant and affects not only early but late vegetables.

The article of Woronin remained for many years the only extensive investigation of this organism. Many notes as to its occurrence, methods of prevention, etc., are to be found in the agricultural literature, but no advance was made in the subject until the publication of Nawaschin.[3] This author undertook an investigation with modern histological methods to clear up the obscure phases of the organism in the plant cells, to investigate the changes produced in the plant by the organism, and the nature of the relations between the cell and the parasite. It may be remarked that the closing paragraph of Woronin's article called attention to a possible relation between these tumors in plants and those found in man and animals. The wording of this paragraph is so significant, in the light of present investigation and it is so seldom referred to, although made in 1878, that it would seem desirable to give it verbatim.

"Ich vermuthe nämlich, dass die Erscheinung und Entwickelung vieler pathologischer Auswüchse und Anschwellungen, die auf dem thierischen Organismus vorkommen, auf folgende Weise arklärt werden können: In den lebendigen Organismus dringen, auf irgend welchem Wege, kleine Myxamöben ein, die sich allmählich in Plasmodien entwickeln und im Gewebe dieses oder jenes Organs eine bedeutende Reizung hervorrufen; dieses verursacht im Organ eine pathologische Veränderung des ganzen Gewebes, von welcher denn auch die Form und Grösse der krankhaften Auswüchse oder Anschwellungen abhängig sind. Nächstfolgende Untersuchungen werden zeigen, ob meine Vermuthung sich bestätigen lässt."

In the introduction to his article Nawaschin calls attention to the closing paragraph of Woronin's article above given and notes the fact that from the standpoint of general pathology, Plasmodiophora is worthy of special attention as an intracellular parasite producing a specific tumor which bears many analogies to the so-called "malignant" tumors in animals and man. He furthermore calls attention to the fact that Plasmodiophora un-

doubtedly stands very close to the *Sporozoa,* the recognized cause of many infections in animals. Nawaschin's work deals exclusively with the results of sectioning and the staining of the plant tumors and has thrown much light on the intracellular phases of the organism. This, he finds, makes its first appearance within the plant cell in the form of a well-developed amœba, which, in osmic acid preparations (Flemming or Hermann), may be immediately detected within the plant cell as a mass of closely packed, regular, blackened, granules. On careful examination with higher powers these granules are found to be embedded in a mass of protoplasm with more or less well-defined outlines (Fig. 4). Between the granules one or more clear spaces will be found within which a spherical, centrally placed body, bright red in safranin preparations, can be distinguished. These prove, on further observation to represent nuclei. If the osmic acid is removed from the preparations these nuclei are found to be surrounded by the protoplasm of the amœbæ. The youngest amœbæ observed by Nawaschin have always possessed at least two nuclei. These present definite characteristics. They are spherical or oval in the resting stage, have a fairly well-developed nuclear membrane, are apparently filled with a clear fluid and possess a highly refractile central body, as he expresses it, "of nucleolar nature." The infected plant cells are always arranged in groups and each cell usually contains more than one amœba.

The normal plant cell possesses a well-defined membrane of cellulose lined by a narrow layer of protoplasm which extends in bridges to the nucleus, which lies in varying positions within the cell cavity. The spaces between the bridges of protoplasm are filled with cell sap. It is within the sap spaces that the amœbæ first make their appearance. The method of their entry as swarm spores into the infected plant is, as we have stated, as yet undetermined. The nucleus of the plant cell is spherical, oval, or of irregular shape, possesses a nuclear membrane and a large, spherical or irregularly shaped nucleolus. It is many times larger than the diameter of the largest nuclei in the amœbæ and can readily be distinguished by its characteristic appearance. The conditions within the plant cell are so simple that the morphology of the parasite and its relation to the cell can be determined with great accuracy. Fig 4 after Nawaschin represents a preparation from which the osmic acid has not been removed and the amœbæ are seen within the plant cell sharply defined by the regular, black granules embedded in their protoplasm. The differentiation of the protoplasm of the amœbæ in preparations from which the osmic acid has been removed, is not

invariably well-defined. This is especially true of the very young organisms. In these the protoplasm of the organism may be poorly developed and in fact, consist of mere threads extending into the surrounding plant protoplasm, in which it is apparently lost. Figs. 5 and 6 from Nawaschin represent two young amœbæ immediately after division of the nuclei. The protoplasm in these cases presents the characteristics just mentioned.

While Woronin believed that the increase in number of amœbæ within certain cells was probably owing to the fact that the organism at this stage passed from one cell to the other, Nawaschin has shown that this interpretation is incorrect. He has never been able to detect any amœba in the act of passing through the wall pits of the cells. He has been able to determine that they increase in number by division, that the presence of the organisms for a long period causes no change in the plant cell; that during this stage *the organism lives in a state of symbiotic relation with the host cell, until through proliferation the number of amœbæ in the plant cell makes sufficient demands upon the nutrition of the cell, whereupon the host cell divides by the regular process of karyokinesis.* The amœbæ in this case are more or less equally distributed in the daughter cells. (See Fig. 7 from Nawaschin.) By this means, according to Nawaschin, the organism is distributed in the diseased portions of the plant. The fact that the infected cells lie in groups is thus explained. When, however, the demand upon the infected cell can no longer be met and the nutrition is finally exhausted, the amœbæ pass through certain preparative changes, finally coalesce to plasmodia, the nuclei all divide by the regular process of karyokinesis and the entire mass is divided up into minute myxamœbæ, each with a single nucleus. These myxamœbæ secrete a membrane about them, take on a spherical form and become condensed into spherical spores. There is no sac (peridium) formed about the plasmodium in this process of spore formation, but, as Woronin has pointed out, in all probability the absence of the peridium is due to the fact that there is no necessity for this formation, the walls of the exhausted plant cell performing this function.

The first evidence of nuclear division is found in the appearance within the clear space between the periphery and central body of minute grains of chromatin irregularly placed. These arrange themselves in a plane which later becomes that of division, in a circle about the central body. This first becomes elongated and then divides. A longitudinal section at this stage shows two closely approximated central bodies with two small grains of chromatin in the plane of division, but close to the

divided central body. In cross section this figure usually presents the appearance of a rosette, the central body being surrounded by more or less equi-distant granules of chromatin. The central bodies then withdraw to the poles of the cell, half of the chromatin becomes intimately associated with each. The elongated nucleus is then divided into two spherical smaller nuclei by the capsule passing into the center and coalescing. In this phase it commonly occurs that the entire elongated nucleus is bent upon itself, which results in the nuclei, when completed, being placed at a slight angle to each other and presenting a characteristic appearance, as shown in Fig. 6 from Nawaschin. Nawaschin has not attempted to analyze this method of nuclear division, but it will be seen that it is entirely distinct from the process of coalescence and regular karyokinetic changes in the resulting plasmodium which terminates in spore formation.

It will be seen by observing the illustrations taken from Nawaschin that the nucleus of the amœboid form of Plasmodiophora presents a very distinct similarity of appearance with the cell inclusions noted in cancer, a fact which has been noted by von Leyden and others.

The analogy between the tumors resulting from the infection of the plant by this organism and malignant tumors in man and animals, as pointed out by Woronin, was emphasized by the observations of Behla,[4] who noted the great frequence of clubroot in the gardens of the inhabitants of a certain portion of the town of Luckau, in which cancer was particularly prevalent. He likewise found that fragments of the plant tumors inoculated into animals produced swellings and tumor-like formations, but he was unable to detect the parasite in the animal tissues. A more direct support of the analogy is to be found in the preliminary statement of Podwyssotzki, 1900, who found that animals inoculated with fragments of clubroot developed tumors which possessed certain points of similarity to the sarcomata and endotheliomata. The attention of Podwyssotzki was attracted to *Plasmodiophora Brassicæ* through the work of Nawaschin, both of these observers being members of the faculty of the University of Kieff. Podwyssotzki's conclusions are of such great interest that it is desirable to quote them in full:

1. It is possible to produce by inoculation with Plasmodiophora Brassicæ, tumors in animals, thus far rabbits and guineapigs.

2. These tumors are of mesodermic origin and in the progressive stage are similar to large celled sarcomata or endotheliomata or the granulomata found in leprosy.

3. These tumors are the result of a marked hypertrophy and proliferation of the fixed connective tissue elements, especially the endothelium of the perivascular lymph spaces. One is, therefore, justified in speaking of a mycetozoan perithelioma or granuloma. The proliferation leads to the formation in the neighborhood of the blood vessels of larger or smaller infiltrations or nodular formations. Leucocytic infiltration is demonstrable in the first stages of the infection but disappears from the eighth to the twelfth day.

4. The spores of *Plasmodiophora Brassicæ* are found in the tumor cells either singly or in great numbers. In the latter case the cells are greatly distended and the spores are surrounded by a mucin-like substance which gives the whole the appearance of a plasmodial mass filling the body of the cell.

5. The more mature tumor cells contain the greater number of spores. Many of these are so greatly distended and show such marked vacuolization that they are apparently being destroyed. For this reason the central portion of the tumor is often represented by a yellowish, half dry mass of disintegrated cells.

6. The most peripheral or youngest elements of the tumor contain fewer of the spores. These are difficult to demonstrate. It can only be accomplished by a careful comparison of the different stages of metamorphosis from the centrally arranged older cells of the tumor containing large numbers of well-defined spores.

7. In many places in these tumors may be found giant cells, including within their bodies numbers of spores. In some of these giant cells the spores have disappeared.

8. The nuclei of the infected cells for a considerable period show no changes aside from increase in size and chromatin content. Many of the cells containing spores show evident mitosis, proving that the parasite induces proliferation of the nuclei.

9. The picture presented in these nodular formations can be most easily understood as evidence of specific phagocytosis, as undoubtedly many of the spores are destroyed within the cells, in which case they gradually become incorporated with the protoplasm of the cell and can no longer be differentiated. It was undoubtedly the cells of this sort which were observed by Behla and which led him to the conclusion that portions of clubroot after transplantation into animals produced infection, although the parasites could not be detected within the cells.

10. It would seem that the nuclear substance of the spore undergoes a progressive metamorphosis. In many of the spores two or more chromatin-containing nucleoli may be found. The

FIG. 1

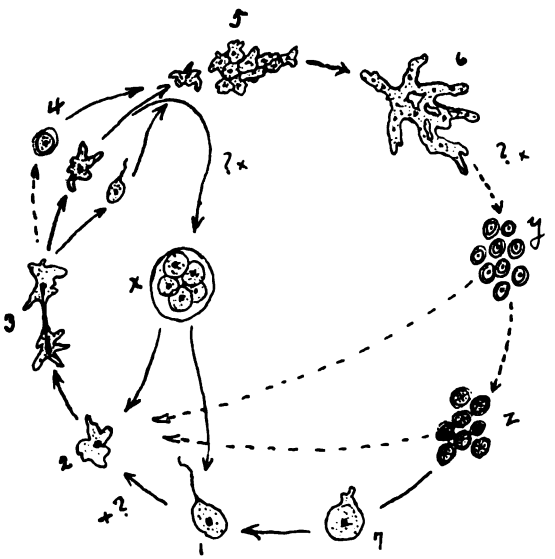

FIG. 2.

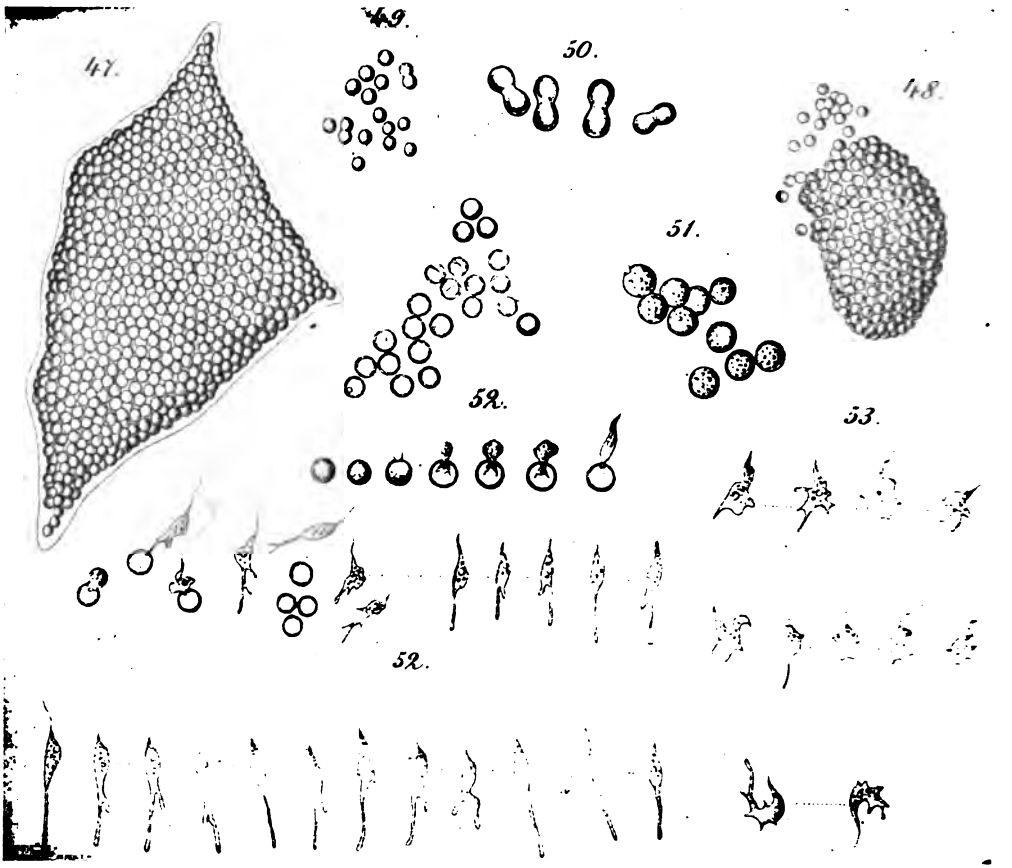

FIG. 3.

Figs. 47, 48, 49, 50, Resting spores; 51, beginning germination; 52, escape of the myxamoebae from the cysts; 53, free amoebae.

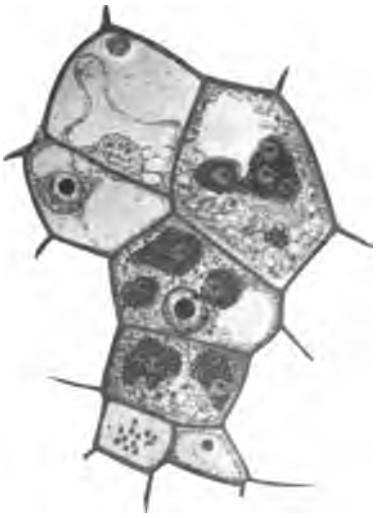

Fig. 4.

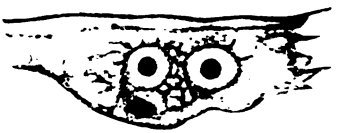

FIG. 5.

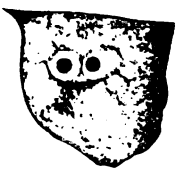

Fig. 6.

spore in this case presents an appearance suggestive of the differentiation found in mitosis. It is, however, impossible to affirm with certainty that an actual increase of elements within the spore occurs. This question must be the subject of future investigation. In many cells, however, the parasite may be found without the characteristic capsule. Under any circumstances it would be difficult to explain the infection of cells so far from the center of the tumor without an increase of elements within the spore, as it is impossible that the comparatively large spores (about the size of an erythrocyte) should be carried by the lymph through the small lymph spaces. Spores within the leucocytes have never been encountered.

11. The larger cells in sections fixed in Flemming's fluid present an extremely interesting picture. Surrounding the single spores the adjacent protoplasm of the tumor cell is filled with closely arranged minute fat droplets, so that the infected cell is readily detected even with low power. Such cells present the appearance of a vacuolated cell each vacuole surrounded by fat droplets. Under oil immersion the structure of the cell can be readily determined and it would appear that the minute fat droplets are a portion of the parasite, the protoplasm of which, in the vegetable tumors, is normally filled with fat droplets. The fat remains within the spores after implantation in the tissues of animals, so long as the spore remains free. It diminishes in the spores so soon as they are taken up by the connective tissue cells. When this has occurred, the spores gradually lose their fat with the exception of one or two minute fat droplets within the capsule.

12. In an extremely malignant case of sarcomatosis of the peritoneum, kidneys, thyroid and other organs of a child from the clinic of Prof. Tchernoff, I have observed the presence of cells presenting the characteristics of those above described. They were especially prominent in preparations hardened in Flemming's fluid where they could readily be detected with low power, because of the blackening resulting from the presence of the fat. Under oil immersion these cells were found to contain a number of minute round bodies within vacuoles. A further report of these parasitic inclusions will appear later.

Since the appearance of the preliminary statement of Podwyssotzki no one has, apparently, disproved or confirmed his results. One or two observers have confirmed the similarity of appearance between certain forms of Plasmodiophora and the inclusions in cancer (von Leyden, Feinberg), while others (Lubarsch, Nösske), believe that this analogy is not justified.

Results of inoculation experiments with Plasmodiophora.— Early in 1901, through the kindness of Prof. Welch, we came into possession of the article of Nawaschin, which naturally fixed our attention upon the preliminary statement of Podwyssotzki, above mentioned. Since that time we have been experimenting with Plasmodiophora in various ways. Through the kindness of Dr. Erwin F. Smith, of the Laboratory of Plant Pathology, Washington, D. C., we were placed in communication with Prof. L. R. Jones and Mr. W. J. Morse of the Agricultural Experiment Station at Burlington, Vermont. These gentlemen have repeatedly furnished us with young clubroots, grown for us in their greenhouses during the winter. Through Dr. Smith we were likewise placed in communication with Dr. M. C. Potter of the Durham College of Science, Newcastle-upon-Tyne, from whom we were able to obtain a quantity of clubroots from England. Careful comparison of the Plasmodiophora obtained from this source and that found in America shows no distinguishable difference. To all of the gentlemen mentioned we are deeply indebted and desire to express our thanks for much assistance.

With the material obtained from these sources and with clubroot obtained from the neighborhood of Brookfield, N. Y., where the disease is prevalent, to which our attention was called by Dr. I. P. Lyon, and the neighboring town of Hamilton, (Fig. 1) where it is apparently still more prevalent, we have had no difficulty in infecting earth. Cabbages and other varieties of Brassicæ grown upon this ground either in the greenhouse or in the open, in a prepared bed at the rear of our laboratory, have supplied us continually with material. Fig. 8 is a photograph of a specimen raised in the bed above mentioned during the past summer. We began our investigations by careful examinations of the fresh material. The spores of the organism could be readily detected in free hand sections, or in scrapings from the fresh surface of the clubroot. Many of the infected cells were found filled with these closely packed spherical bodies. (See Figs. 3-43 from Woronin.) They measured 3.33 μ in diameter. Some of them were homogeneous (see Figs. 3-49 from Woronin), in some a delicate outline within the delimiting capsule could be observed and in the majority one or more highly refractive bodies. *The relation of these spores to osmic acid was ascertained with the result that the various forms were found to blacken after treatment of a few hours. This blackening affected the capsule of the spore and the highly refractive central body. Where the spores were homogeneous they took on diffuse blackening.* The color was usually yellowish green, rarely of a

brownish tinge. *When treated with Sudan III, some of the spores stained intensely while others took up the stain less rapidly. In the spore forms containing a well-defined, highly refractive central body, this and the capsule were usually first blackened by osmic acid or stained by Sudan III, as the case may be.*

In young clubroots the amœbæ described by Nawaschin could be readily detected. These were found in the form of spherical or oval masses of protoplasm within the sap spaces of the plant cell, filled with coarse granules, approximately one micromillimeter in diameter. These granules were highly refractive and in many cases were in active motion. The nuclei of the amœbæ were visible as vacuoles in the protoplasm and occasionally a highly refractive central body could be observed. The whole appearance of these forms *with the exception of the nucleus* could only be compared to that of an *eosinophile*, as they appear in fresh preparations. The striking character of this resemblance was extremely impressive. Occasionally free amœbæ could be detected in the fluid of the scrapings, in which case they were spherical, the granules usually in active motion. Attempts were made to fix and stain them but without great success, the best results being obtained in the sections to be described later.*

For the purpose of observing the development of the organism, portions of dried clubroot were placed in flasks containing sterile water and allowed to remain at room temperature. In many cases the water in the flasks was rapidly overgrown by bacteria, but in a few cases where the bacteria did not develop with such rapidity, we were able to trace the development of the swarmers within the spore cysts, their liberation from the spore cysts and their presence free in the fluid. The smallest recognizable form of the organism is a spherical, highly refractive body resembling a minute drop of fat, varying in size between 1 and 3 μ, *each having attached to it a delicate, thread-like flagellum, so fine as to be scarcely distinguishable even with the highest powers. This minute, spherical form of the organism moves about rapidly in the field by the movement of this delicate thread. In preparations treated with osmic acid we were able to determine that the body of this form of the organism fails to blacken even after prolonged treatment.* The next recog-

* This phase of the organism will continue to offer a field for research for some time. The observations thus far reported are largely confirmatory, with the exception of certain forms thus far undescribed. An article on the staining affinities of various forms of the organism will be published shortly by Dr. Matzinger, with whom the observations on the ectogenous cycle of Plasmo-diophora Brassicæ were jointly conducted.

nizable form of the organism are the myxamœbæ and myxoflagellates, the latter being more common. The swarmers can be seen developing within the spore cyst, as described by Woronin (see Figs. 3, 51, 52). It was likewise possible to observe the exit of the swarmers from the spore cysts, as illustrated in Fig. 9, microphotograph.

Our experience in this connection is of interest, inasmuch as no less an authority than Tubeuf[5] has recently stated, that he has never been able to observe this phase and that the late Prof. Frank had likewise never succeeded in causing the spores to develop into cysts with the liberation of the swarmers. Von Leyden has also made the same statement.

The development of the swarmers within the spores begins by the spore increasing in size to about 4 to 6 μ, the highly refractive body within the spore sac gradually disappearing and the contents of the sac taking on a well-defined granular character. This granular protoplasm (also shown by Woronin) gradually contracts until the mass within the spore sac can be easily distinguished from the surrounding membrane. At the proper time the spore sac bursts, liberating the mass of protoplasm, which, after passing into the surrounding medium takes on the form of a myxamœba or of a myxoflagellate. The protoplasm of the organism at the time of liberation is homogeneous. A nucleus can occasionally be observed but is extremely indistinct. All attempts to harden and stain the organisms in any of the forms described, have given but unsatisfactory results. In the most successful attempts the organism appears as a mass of protoplasm of rounded or irregular form with a center staining somewhat more deeply than the surrounding protoplasm. We have never been able to demonstrate the presence of a contractile vacuole. The organism in this stage is so lacking in characteristics that all attempts to establish a means of identification have been unsuccessful.

Inasmuch as it is in this phase that the organism is supposed to infect the plant, we have attempted to detect the swarmers and flagellates in the tissues of animals. Cultures containing large numbers of the swarmers have been injected into animals intravenously and directly into the tissue, the animal being killed and the tissues hardened within a few minutes after the injection. In no case have we definitely succeeded in locating the swarmers by their appearance. *Once in the tissue, becoming rounded, they present an appearance so like structures usually interpreted as degeneration products that it is impossible at present to recognize the organism with certainty.*

Examinations of clubroot by means of hardened and stained

FIG. 7

FIG. 8

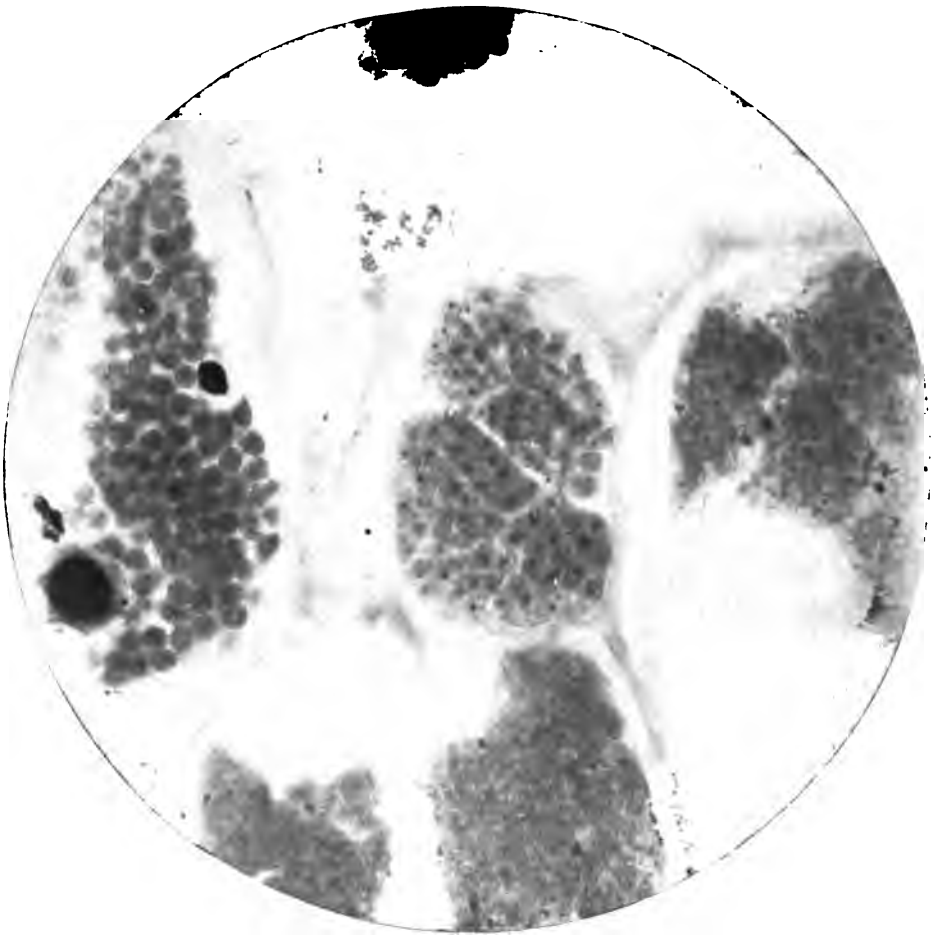

Fig. 9

sections.—The staining method which we have principally utilized is the method of Plimmer, inasmuch as we desired from the beginning to compare the appearances of the organism to the cell inclusions in cancer, as demonstrated by this method. The method recommended by Nawaschin has likewise been employed, and, as given by this author, is as follows: Very small portions of the infected root are placed in strong Flemming's solution, where they are allowed to remain twenty to twenty-four hours, when they should be removed, as longer action of the fixative permanently blackens the protoplasm of the organism. After embedding in paraffin, sections two or three micromillimeters are cut and attached to the slide by cohesion. When it is desired to remove the osmic acid from the preparation the sections and slides are placed in hydrogen peroxide full strength. Preparations are mounted in thickened cedar oil, stained with Heinberg's safranin-gentian-violet-orange stain. Sections are first stained with one per cent. safranin solution, washed in water and then counterstained with two per cent gentian-violet solution. Pass through alcohol and clear in clove oil which has been saturated with orange, in which they may remain a considerable length of time without overstaining. They may then be cleared with xylol and mounted in balsam, or cleared in commercial oil of cedar and mounted in thickened cedar oil.

This method gives excellent preparations but in no way superior to those obtained by the method recommended by Plimmer for the demonstration of cellular inclusions in cancer.

Sections of mature clubroots are distinguished by groups of hypertrophic cells filled with closely packed spores. Preparations hardened in Hermann and stained with safranin show that many of the spores are blackened by the osmic acid. Those which do not show the black reaction stain diffusely with the safranin. The dense delimiting membrane in the unblackened spore can be detected by a slight difference in the refractive index. Within this an irregular outline, possibly representing the protoplasmic content of the spore, is distinctly visible. The spores present a most interesting appearance in preparations from which the osmic acid has been removed and then treated according to Plimmer's method. In this case it has been found that the reaction of the spore to the iron hæmatoxylin is almost as capricious as its relation to osmic. Many of the spores are stained a diffuse blue-black, in which case the internal structures are obscured. A certain number are entirely resistant to the nuclear stain, in which case the irregular internal structure can be readily observed. (Fig. 9.) The spores are of uniform size and measure, as above stated, 3.33 μ. The amœbæ

present the characteristics already given by Nawaschin. In the younger roots examined they are the predominating form and *in the very young outgrowths the spores are entirely absent.* The nuclei of the amœbæ stain with a protoplasmic stain, Plimmer's method, thus presenting an appearance extremely like that of the so-called "Plimmer's body," the characteristic cell inclusion of cancer. They vary considerably in size in the different amœbæ but the nuclei in each amœba are of practically the same dimensions. (Fig. 10.) We have detected nuclei as small as two micromillimeters in diameter and others five micromillimeters. Most of the nuclei are spherical in outline, but many oval or fusiform nuclei will be encountered. The protoplasm of the amœbæ in osmic acid preparations is either granular or coarsely reticulated, depending upon the success of the fixation. The outlines of the mature amœbæ can be readily determined *but in the younger forms, as pointed out by Nawaschin, the protoplasm is intimately associated with the protoplasm of the plant cell and is often poorly developed, in which case the nucleus presents an appearance extremely like the cell inclusions in cancer. The central body stains somewhat more intensely than the nuclear membrane. The nuclear membrane of the smaller nuclei is, as a rule, poorly developed in many cases scarcely distinguishable, in which case the nucleus presents the appearance of a vacuole in the protoplasm containing a highly refractive central body.* Many of the cells are completely filled with closely packed amœbæ in which case the cell protoplasm is practically obliterated. The infected cells lie in groups, as described by Nawaschin, but these groups are usually surrounded by hypertrophic plant cells containing no parasites. The nuclei of the plant cells are readily distinguishable from the amœbæ, measuring on the average ten to twelve micromillimeters in diameter. The amœbæ vary in size, the smallest measuring four or five micromillimeters, the larger eighteen or twenty micromillimeters in their greatest diameter. The form of the amœbæ varies within wide limits but the predominating form is oval or spherical. We have counted as many as twenty nuclei in one section of an amœba.

In the material thus far examined we have been unable to collect all the different forms described by Nawaschin, as evidence of nuclear division of the amœbæ, but divided central bodies with the grains of chromatin arranged in the plane of division are not infrequent. For this purpose young roots are required. The phases leading to spore formation are more readily discernible. Large amœbæ in which the nuclei are indistinct are extremely frequent. The outline of the amœbæ in this stage becomes indistinct (Fig. 9) and those adjacent to

each other coalesce. Many of the cells are filled with granular masses of protoplasm nearly or completely filling the entire cell (plasmodia). (Fig. 9.) The nuclei of these masses of protoplasm, which are scarcely discernible, then divide by a process of regular karyokinesis, so that large masses are found filled with spindles, with well-defined medullary plates. At this stage the entire mass of protoplasm divides into irregularly shaped units surrounding the spindles, and these, according to Nawaschin, by a process of condensation, form the spherical spores.

Infection of animals with Plasmodiophora.—Having determined the appearance of the spores, amœboid and plasmodial forms of the organism within the plant, both in the fresh state and in hardened sections, we have undertaken to repeat the experiments of Behla and Podwyssotzki, by implanting fragments of the tumor-like outgrowths of the infected roots beneath the subcutaneous tissue and into the peritoneal cavities of cold- and warm-blooded animals. For this purpose we have employed guinea-pigs, rabbits, a few white rats and frogs.

The method of procedure was as follows: Fresh clubroot immediately after removal from the ground was washed for a period of time in running water, then carefully scrubbed with a brush to remove the dirt, after which it was thoroughly scrubbed in a 1:2000 sublimate solution, then thoroughly washed in sterile water. Observing the usual antiseptic precautions, sections of the tumor-like outgrowths eight or ten micromillimeters in thickness were cut from the root and with a sterile canula disks were punched from the cortical portion of the outgrowth, in each case avoiding the periphery. A slight incision was made through the skin in guinea-pigs and rabbits, the canula was introduced, the disk of clubroot pushed from the canula into the integument and the wound closed.

In all of the animals thus treated the wound healed rapidly without suppuration except in a few cases where the clubroot was not sufficiently disinfected and bacteria were introduced. Where infection did not occur, the results were uniform, whether the implantation was made in guinea-pigs or rabbits. In each case microscopic examination was made of the fresh material before implantation. Several animals were inoculated with very young outgrowths, the tissue of which, when examined with the microscope, showed only the presence of amœbæ within the plant cells, and in these cases *no infection resulted.* In the remaining experiments the microscopic examination showed the presence of many spores of the organism, which were likewise readily de-

tected in the fragments of plant tissue remaining after removal from the animal at the termination of the experiment.

In all of these experiments there developed at the site of implantation a distinct tumor, which increased in size for a period of from four to six weeks, at which time they were usually the size of a hazelnut. From the sixth week on the tumors gradually decreased in size and at the end of three months the site of the implantation was marked only by a small mass of connective tissue or had totally disappeared. The microscopic appearance of the fresh tumors during the stage of progression, *i. e.*, to six weeks, was found on removal to consist of the remains of the implanted vegetable tissue surrounded by pinkish-white granulation tissue constituting the tumor. Scrapings from the cut surface of the tumor, avoiding the plant tissue, showed that the growth is composed of large cells of connective tissue of endothelial origin. These cells possess large vesicular nuclei containing one or more nucleoli. An occasional cell is met in which the nucleus is in some stage of karyokinesis. These large cells can be readily detected by numbers of minute, highly refractive fat droplets embedded in the protoplasm. Under high power, besides the nucleus the large phagocytic elements are found to contain within their protoplasm numbers of cell inclusions presenting the appearance of vacuoles containing central bodies. Each cell usually contains several of these inclusions and in some cases the protoplasm is closely packed with the inclusions and fat droplets. Numbers of the inclusions present the appearance of the spores of Plasmodiophora when examined in the fresh state, *i. e.*, spherical, greenish-yellow bodies of high refractive index, with a highly refractive central body. The derivation of the inclusions first mentioned from the typical spores of the organism can be readily traced. In the younger tumors the fresh scrapings show the presence of large numbers of leucocytes. Careful examination of the leucocytes shows that spores or inclusions within the leucocytes are extremely rare. In only one case have we found a spore within a leucocyte.

Sections through the surrounding tissue and the implanted vegetable tissue were hardened in sublimate, formalin and Hermann's fluid. Preparations hardened in Hermann and stained with Plimmer's method gave the most satisfactory results. An examination of growths of ten days or under reveals the following relations. The outline of the plant structure is well preserved and the relations of the contained spores and the remains of the amœbæ can be easily determined. At the margins, the plant tissue is surrounded by a mass of coarse granulation tissue, more or less densely mixed with polymorph nuclear

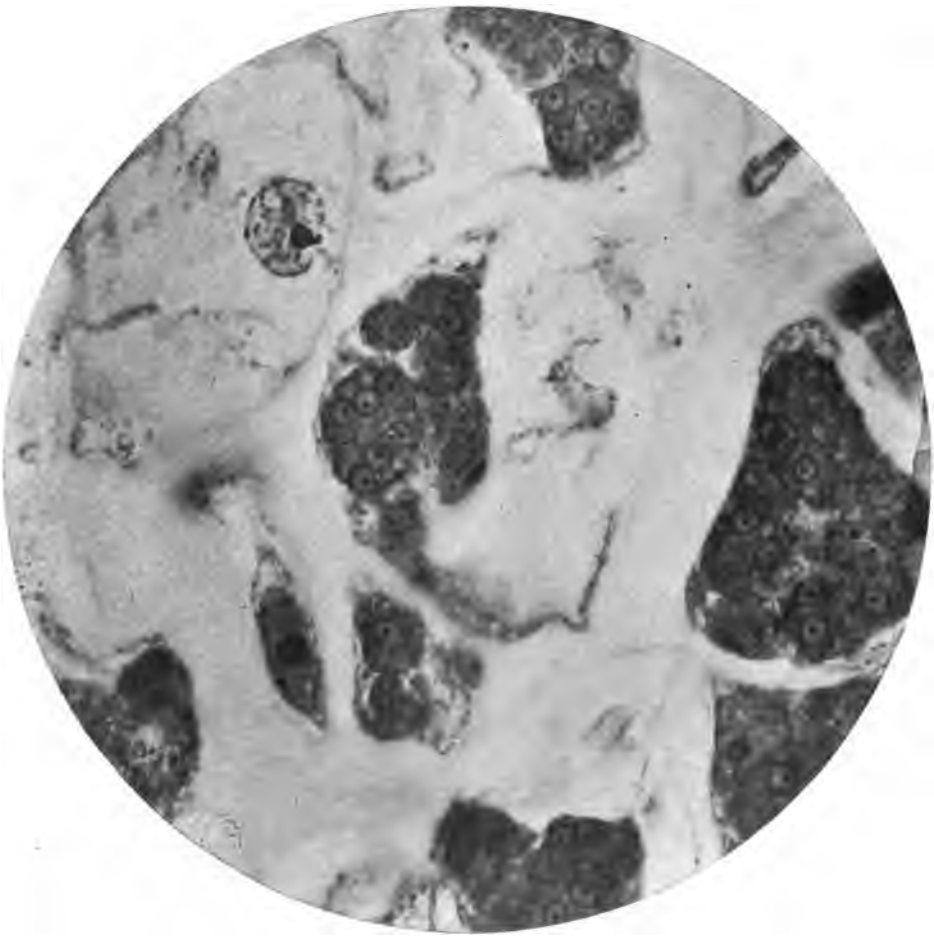

FIG. 10

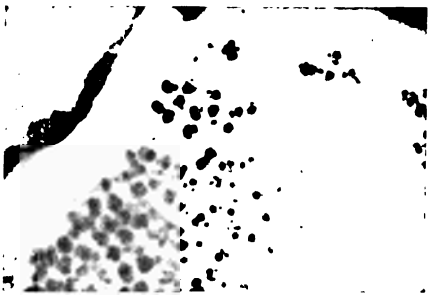

FIG. 11

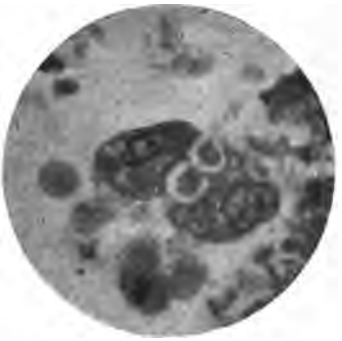

FIG 12

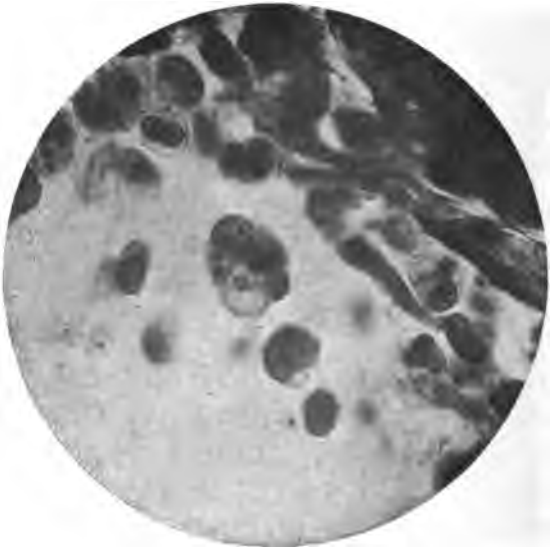

FIG. 13

Fig. 14

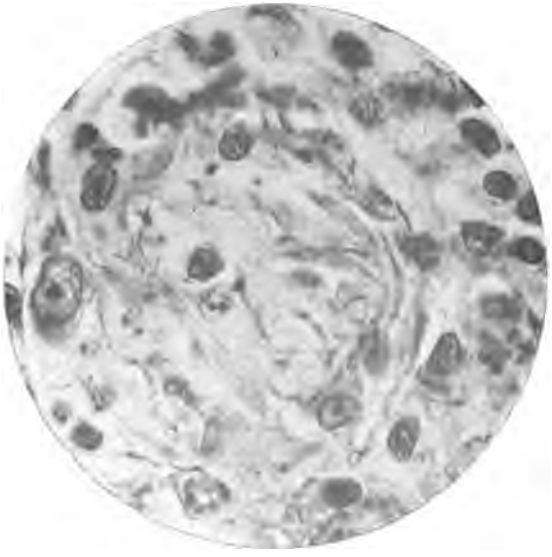

Fig. 15

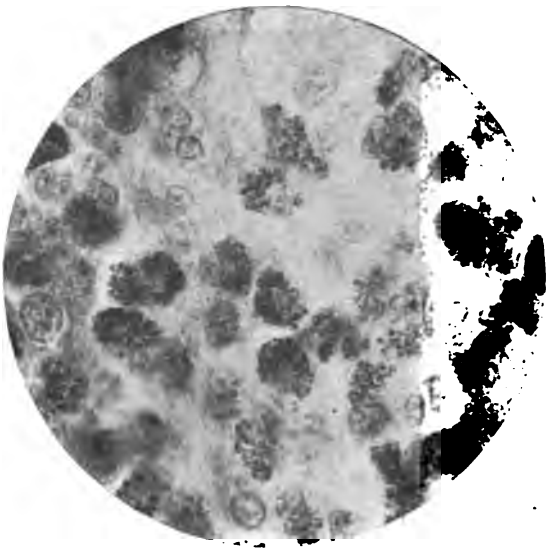

Fig. 16

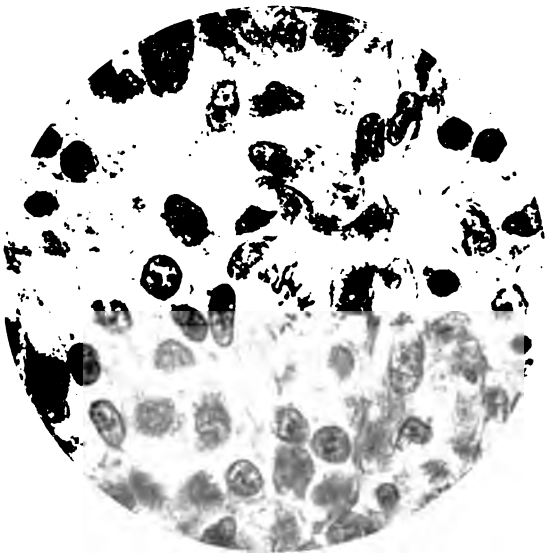

Fig. 17

leucocytes. In uninfected cases the leucocytes are seldom present after the twelfth day, as pointed out by Podwyssotzki. An examination of the plant tissue reveals the spores intact within the infected cells. In preparations from which the osmic acid has not been removed, the spore presents a characteristic appearance. Instead of the diffused blackening which characterizes the spore in the fresh state, in many spores the fat is gathered together in globules at the center. (See Fig. 11.) At the margin of the plant tissue the spores can be traced for a certain distance into the granulation tissue by the blackened droplets of fat which they contain. It is evident by studying the margin of the preparation that the spores rapidly lose their fat, whereupon they can no longer be distinguished in preparations stained with safranin. In preparations from which the osmic acid has been removed and the section stained with iron hæmatoxylin and neutral red (Plimmer's method), the chromatin of the spores stains a blue-black and in the spores remaining in the plant tissue is irregularly arranged near the center of the spore. At the margins of the plant tissue the spores can be detected in the elements of the granulation tissue surrounding it. This tissue is made up of large connective tissue phagocytes with vesicular nuclei of varying size. The nuclei stain deeply and contain one or more nucleoli. The protoplasm is finely granular. In these cells the spores of the organism can be demonstrated, although in preparations from the tenth day on, many show marked alterations in appearance. In early implantations the spores are readily recognizable, still containing chromatin and presenting the appearance found in the spores included in the plant tissue. The large phagocytic cells contain usually one or more, in some cases the protoplasm is filled with closely packed spores. In tumors from the twelfth day on, many of the spores within the cells are found to have increased in size, the chromatin is usually absent, the protoplasm is contracted in the form of a central body surrounded by a clear space (Figs. 12 and 13). The periphery of the spore is indistinguishable from the protoplasm of the cell containing it and the whole presents an appearance closely resembling certain cell inclusions in cancer.

The condition of the amœbæ within the plant tissue after implantation in the subcutaneous connective tissue of animals is worthy of especial attention. *In all preparations we have found that the amœbæ and all forms of the parasite, with the exception of the spores, have disintegrated.* The site of these forms is marked by masses of disintegrated protoplasm and chromatic material. By a careful comparison of portions of the plant tissue

in each case before implantation, we have been able to determine that this is actually what occurs. The infection of the surrounding tissues is therefore owing to the presence of the spores and the amœbæ are undoubtedly incapable of producing infection. This is shown by the cases in which we implanted fragments of young clubroot in which the organism had not reached the stage of spore formation. In all of these experiments the fragment of plant tissue acted simply as a foreign body in the tissue and after the usual period disintegrated and was removed. Macroscopically these cases could be readily distinguished from those in which the plant tissue contained spores, in which case granulation tumors the size of a hazelnut invariably developed. It is *highly probable in this case that the amœbæ perished with the plant cell, in which case the symbiosis referred to by Nawaschin is apparently complete.*

The nodules which result from the infection of warm-blooded animals by the spores of Plasmodiophora disappear in a period varying from six weeks to three months. They are therefore not malignant, the organism being ultimately destroyed by the activity of the cells in which they are found.

Our second series of experiments, in which we employed frogs, presented even more definite characteristics than those found in warm-blooded animals. In these experiments the necessity for preventing the introduction of bacteria was not as great as in the warm-blooded animals. The frog is apparently capable of overcoming bacterial infection with readiness. In these experiments the clubroot was ground in a mortar in sterile salt solution. After determining that spores were present in the material it was strained through cheesecloth to remove the coarse fragments of plant, and then injected into the peritoneal cavity with a hypodermic syringe. Frogs examined at the end of seven to ten days show upon the surface of the peritoneum especially in the omentum, whitish, firm nodules, varying in size from a pinhead to a grain of rice and larger. In some cases the omentum is thickly infiltrated with larger and smaller nodules. Teased preparations from these nodules and scrapings show that they are composed of large endothelial cells containing spores of Plasmodiophora and in scrapings made from the periphery of the nodules large numbers of coarsely granulated elements can be found. In frogs killed after the fifteenth day the granulated cells are greatly decreased in number. The granulated cells were never found to contain the spores of the organisms. The nodules were hardened in formalin, sublimate and Hermann's fluid. Preparations hardened in sublimate and Hermann were best suited for the investigation of the spores

which in the formalin preparations were scarcely distinguishable. In sections hardened in sublimate and stained with hæmatoxylin-eosin the structure of the nodules is well-defined. It consists of accumulations of large cells with large vesicular nuclei of varying shapes. Many of the cells are oval, the majority are fusiform (Fig. 14). They are closely packed and in many cases arranged about centers presenting an appearance not unlike the pseudo-pearls or whorls found in certain endotheliomata (Fig. 15). The nuclei are rich in chromatin, the cell protoplasm stains readily with eosin. On the surface of the smaller nodules which lie directly beneath the peritoneum the superficial endothelium can be readily distinguished from the cells of the nodules. The nodules frequently lie in the neighborhood of blood vessels and from their relation have apparently developed in the sub-peritoneal lymph spaces. About the margins of the nodules the connective tissue stroma is densely infiltrated with polynuclear eosinophiles (Fig. 16). The cells forming the nodules can be readily distinguished in the connective tissue cells of the stroma. At the margins of the larger nodules the cells forming them penetrate into the surrounding stroma in which they form cell nests sharply delimited from the surrounding connective tissue. Frequent karyokinetic figures are found within the nodules which have never been detected in the surrounding connective tissue (Figs. 17 and 18). In preparations stained with hæmatoxylin-eosin, the spores of Plasmodiophora are scarcely discernible. The protoplasm of the cells at the centers of the nodules presents a coarse granular appearance and is apparently filled with rounded bodies of indefinite form, which occasionally stain a pale rose tinge with hæmatoxylin. As will be seen by the further examination of preparations, these bodies are the spores of Plasmodiophora. In formalin preparations the spores are still more poorly defined and would undoubtedly escape notice. It is probable that by the use of this fixative Behla failed to detect the spores of the organism in infected cells.

In sections from material hardened in osmic acid, from which the osmic acid has not been removed, counterstained with safranin, the spores of the organism within the cells of the nodules can readily be detected as blackened, spherical bodies, approximately three micromillimeters in diameter. Under low power (see Fig. 19) the spores present the appearance of fat droplets blackened in the usual way by the osmic acid within the cells. They are most densely arranged at the centers of the nodules, the cells at the periphery containing a fewer number of spores than those forming the center. Under high power (Fig. 20) the

fat is found to be most densely arranged about the periphery of the spore and many spores are found which apparently contain no fat whatever. These shadowy forms, many of which apparently contain no internal structure, look like minute shadowy red blood corpuscles. In preparations hardened in sublimate and stained with Plimmer's method the picture presented is similar to that found in the hæmatoxylin-eosin preparations, with the exception that the nuclei are more sharply defined and many of the spores are found to contain chromatin. The differentiation of the cells of the nodules from the subperitoneal connective tissue stroma is more sharply defined. Karyokinetic figures are likewise more pregnantly stained and are found to be quite numerous. The characteristic nest-like arrangement of the endothelial cells is likewise clearly defined. (See Fig. 14). The granules of the eosinophiles in these preparations are capriciously stained, in many cells unstained, in others distinctly blue. The finer structures of the spores are best observed in preparations hardened in Hermann's fluid and stained with iron hæmatoxylin and neutral red (Fig. 21). In these sections it can be seen that many of the spores have lost their chromatin, while the majority possess an irregular chromatic structure at the center of the spore separated from the delimiting capsule by a narrow space. Many of the spores are entirely free from chromatin and in some the chromatin is arranged about the periphery of the spores in the form of fine granules of uniform size. In a few cells we have seen structures of approximately five micromillimeters in diameter sharply defined from the protoplasm of the cell, containing uniform granules, staining deeply with the nuclear stain. These forms are rare and we believe them to be derived from the spores. Whether they represent a stage of further development or retrograde changes in the spores, it is impossible to determine. Free chromatin granules of this type are likewise occasionally encountered, especially about the peripheries of the nodules. Occasionally spores are encountered which have increased in size, which are free from chromatic material, with an irregular, or spherical central body, again presenting an appearance closely resembling the cell inclusions of carcinoma. The granules of the granulated cells in these sections stain deeply with the iron hæmatoxylin. It is difficult to determine, in many cases, whether one is dealing with the minute spherical elements apparently derived from the spores, or free granules from these cells. In regions in which the granulated cells are abundant the differentiation is often impossible. A few sublimate sections were stained with Biondi-Heidenhain for the purpose of determining the

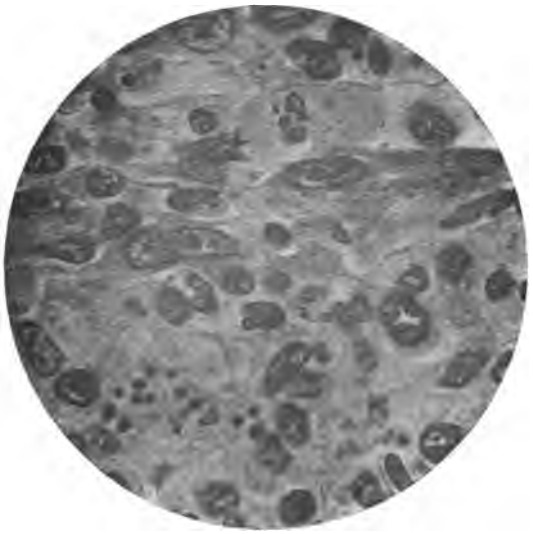

Fig. 18

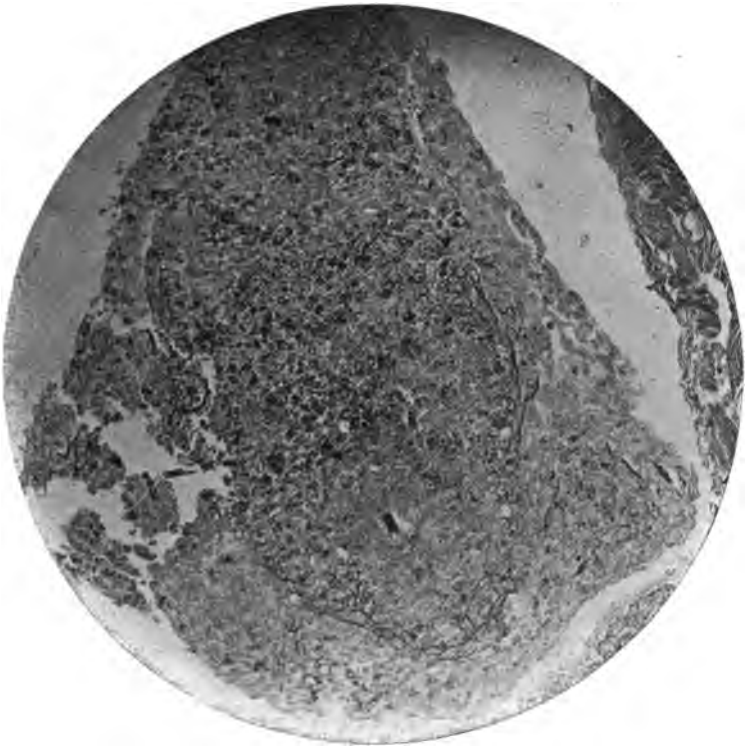

Fig. 19

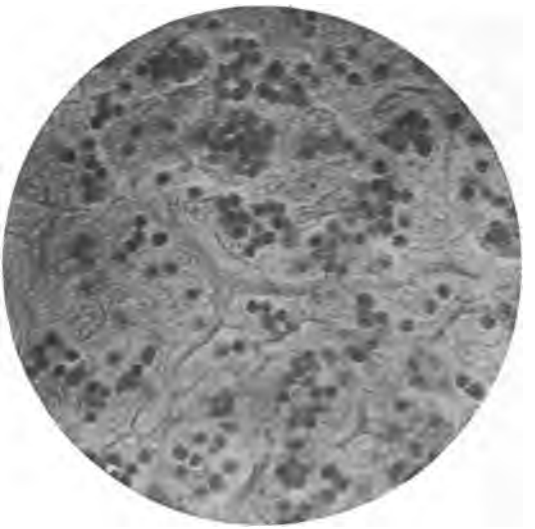

Fig. 20

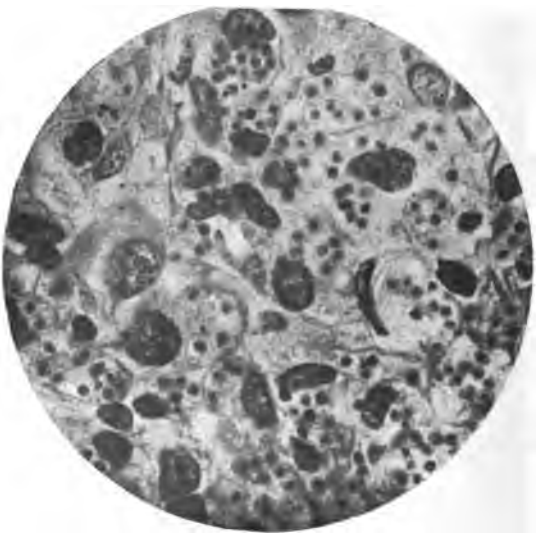

Fig. 21

affinity of the granules of these cells, which were found to stain a brilliant orange. A certain number of the larger endothelial cells near the centers of the nodules were found to contain great numbers of spores. In many cases their protoplasm was greatly distended and the spores were closely packed in large vacuoles presenting an appearance not unlike a cyst filled with spores. Fig. 22 represents a cell of this description from a section stained with Biondi-Heidenhain. The appearance of the spores in these cells is so like illustrations in the recent publication of von Leyden of inclusions found in carcinoma of the lung, that the two should be viewed together (Fig. 23). The tendency of the cells of the nodules to coalesce often leads to the formation of giant cells. In one of our preparations giant cells with the spores arranged about the nuclei were occasionally encountered. The relation of the spores to the nuclei of the cells is often of interest. *In many cases, especially in the smaller cells composing the nodules, the nucleus of the cell will be found to be pushed to one side and curved about the spore, giving it an appearance closely representing certain illustrations of centrosomes.* In the giant cells, the spores are commonly arranged about the nuclei, the center of the giant cell being free (see Fig. 24). In sections stained with Mallory's connective tissue stain, the cells of the nodules stain a brilliant red or orange, whereas the connective tissue stroma stains a brilliant blue. Fine fibres of connective tissue can be found extending into the smaller nodules, but the larger nodules are, for the most port, free from stroma, except about the margins where the cells of the nodules infiltrate the surrounding connective tissue structure from which they were sharply defined.

Frog III killed after forty-seven days, shows upon the surface of the liver, the mesentery and omentum nodules varying from the size of a pinhead to that of a large grain of rice. These are firm and white and of a little greater consistence than those found in Frog I. Fresh examination shows these nodules to be composed for the most part of large, spindle elements in which an occasional vesicular structure varying in size from three to six micromillimeters, can be found. Many of these vesicular structures contain central bodies which are undoubtedly spores of Plasmodiophora. In sections hardened in sublimate and stained with hæmatoxylin-eosin, the tissue of the nodule is found to be composed of large spindle elements with vesicular nuclei. Viewed under low power these cells are seen to be arranged about centers presenting an appearance like the whorls of cells found in certain sarcomata. Occasional giant cells are present measuring as high as thirty micromillimeters in greatest diameter.

These contain many nuclei, as many as eighteen or twenty being counted in one cell. These are usually arranged about the periphery of the cell or in a group at the center. Careful examination of the preparation for spores shows that occasional cells contain shadowy spherical bodies usually without other structure, but occasionally with a central body. Many of these spores are as large as six micromillimeters and present the characteristics of intracellular inclusions found in carcinoma. Occasionally a granulated cell can be found at the margin of the nodules. The cells of the nodules do not stain as deeply as those of Frog I and the intracellular connective tissue stroma is more prominent. Embedded in one of these nodules are two cystic structures of spherical outline measuring eight micromillimeters in diameter. Their margins present a beaded or radiating appearance and each contains a mass of unstained protoplasm of irregular form nearly filling the cyst cavity. The appearance of these structures is so striking as to leave absolutely no doubt that they *represent the spore cysts of Plasmodiophora with well developed swarm spores within them.*

From these experiments it will be seen that the spores of Plasmodiophora when injected into animals, are capable of causing granulomata composed largely of endothelial cells which present certain characteristics distinguishing them from the ordinary infectious granulomata and which give them an appearance more closely resembling endothelioma and sarcoma than the granulomata usually encountered. From a careful survey of our preparations we are able to confirm the conclusions of Podwyssotzki given on page 359 and further to add that the observation in Frog III of perfectly developed spore cysts inclosing what seem to be well developed swarmers shows that the organism is capable of development to this stage within the tissues of cold-blooded animals. These nodules, as stated by Podwyssotzki ultimately subside and resemble endotheliomata only in their histological characteristics. The spores, both in warm and cold-blooded animals undergo certain alterations in the tissue, after which they closely resemble certain of the bird's eye inclusions in malignant tumors.

The large numbers of eosinophiles noted about and in the nodules in the animals infected with spores of Plasmodiophora calls to mind the similar accumulations found in the lymphomata of Hodgkins' disease. The spores of Plasmodiophora in the tissues of both warm and cold-blooded animals correspond in size and appearance very closely to certain structures described by Flexner[6] in lympho-sarcoma which he suggested were protozoa. It is possible that a further investigation of Plas-

modiophora and allied organisms may throw light on the nature of the spherical bodies described by the above author.

The analogy between clubroot and cancer.—The application of the facts pertaining to the distribution of the amœboid form of Plasmodiophora in clubroot, as determined by Nawaschin, to the nature of the proliferation of the cancerous epithelium as it is now understood in man, reveals the following status: The outgrowths of clubroot are the result of proliferation and hypertrophy of the cells of the cortex. The cells of the medullary portion of the plant are never infected. Inasmuch as the infection of the plant can occur during any stage of its development, this must be attributed to a selective faculty on the part of the swarm spores at the time of their entry into the plant tissue. Examination of sections of young clubroot in which the amœboid is the predominating form of the organism discloses the characteristic arrangement of the infected cells as pointed out by Nawaschin. A similar distribution can be found in the beautiful illustrations of Woronin (Fig. 25). The infected cells are in every case arranged in groups and serial sections show that each infected cell is at some point adjacent to another infected cell, the whole group surrounded by hypertrophic cells in which no parasites are to be found. This peculiar arrangement of the cells and the fact that the amœbæ have never been detected in the act of penetrating the wall pits of the cells, has led Nawaschin to the conclusion that the *distribution of the parasites is owing exclusively to the proliferation of one or more primarily infected cells.* That he has found dividing plant cells containing amœbæ and that the amœbæ are in this way distributed, is beyond doubt. The distribution of the groups of infected cells throughout the cortex is undoubtedly the result of multiple infection. An examination of the hypertrophic cells of the cortex surrounding the groups of infected cells, but free from parasites, reveals the presence of an occasional karyokinetic figure. From this it would seem that the amœbæ, themselves incapable of penetrating from one cell to another, liberate some soluble toxic substance which, diffusing into the surrounding cells of the cortex, leads to hypertrophy and occasionally to proliferation. This toxic agent, whatever its nature, is likewise capable of causing proliferation of the cells forming the vessels of the plant, which in some cases proliferate to such an extent that in the neighborhood of the infected cells they are frequently tortuous, coiled and otherwise distorted. This condition is admirably shown in Fig. 26 from Woronin. In no case have parasites been found within the cells of the vessels. On examination of sections of clubroot in which the amœbæ have

passed through the plasmodial phase terminating in spore formation, it will be noted that the hypertrophic character of the surrounding parenchyma cells has largely disappeared. It would thus seem that the toxic action of the parasite is confined to the vegetative stage. The proliferation of the cells in the tumor can therefore be divided into two classes, first, infected cells; the stimulus for the division in this case is probably toxic but the proliferation is no doubt for the purpose of throwing off the parasite, which is seldom successful, as the amœbæ proliferate much more rapidly than the host cell, and by the time the process of proliferation is terminated in the plant element, the entire cell being filled with amœbæ, a certain number remain in each of the daughter cells formed by the division. The second order of proliferation is that affecting the surrounding structures. This is purely toxic and appears to subside when the proliferative phase of the organism terminates. This affects the adjacent cells of the cortex and the cells of the vascular system. If fragments of clubroot containing amœbæ are transplanted upon the cortex of an uninfected plant of the same variety, a tumor will develop which is the result of proliferation of the infected transplanted cells, combined with hypertrophy and a certain amount of proliferation in the surrounding parenchyma cells of the plant upon which the infected cells have been implanted. There is no doubt whatever, if cells of the tumor in the first case could be selected which contained no parasites, no tumor formation would result in the inoculated plant. It will thus be seen that in order to produce clubroot by inoculation, infected cells must be transplanted. In this case, likewise, both cell and parasite must be transplanted intact. Attempts to produce inoculation tumors in plants with the amœbæ alone have thus far proved negative and so far as our present knowledge extends, a primary infection can only be induced by the introduction of the flagellate form of the organism after the germination of the spores in the earth. The endothelial connective tissue tumors which result in animals from the implantation or inoculation of the spore form of Plasmodiophora from the plant are probably largely the result of toxic substances contained within the spore, acting upon the cells of the animal. The transient leucocytosis likewise strongly suggests this explanation. As the organism is incapable of accomplishing a reproductive cycle when implanted under these conditions, the process is not progressive, although the spores are found shortly after implantation embedded in the cells which have proliferated about them.

The frequent occurrence of karyokinetic figures in the cells of the animal forming these tumors indicates that the mere pres-

Fig. 22

Fig. 23

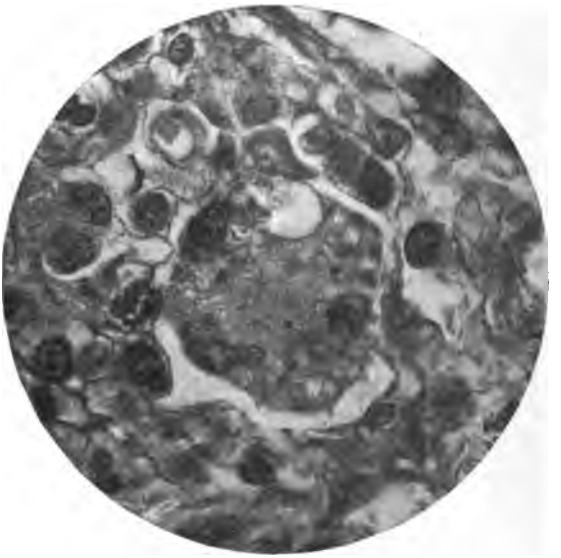

FIG. 24

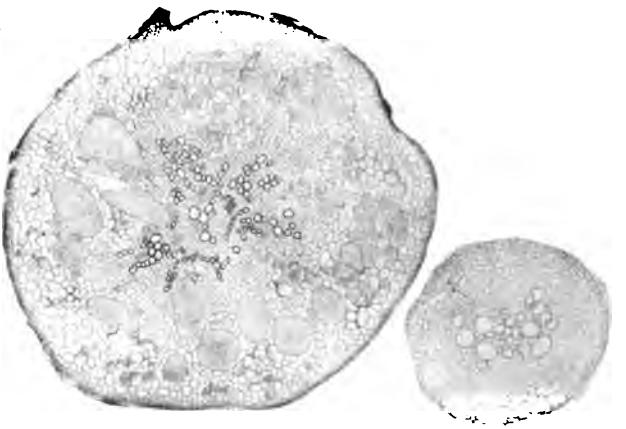

FIG. 25
Infected

Normal

FIG. 26
After Uoronin

ence of the spores is capable of inducing proliferation of certain elements in the animal. It must be noted that this toxic activity of the spores is exerted in a selective way. The cells surrounding the implanted spores, with the exception of the primary, transient leucocytosis, are all of one character and apparently derived from one source. Instead of a heterogeneous mixture of elements such as we find in granulation tissue, these nodules are all of the same type and in the frog present an appearance in the progressive stage closely resembling endothelioma.

If, in the light of the ascertained facts governing the relation between Plasmodiophora and the plant cell, as above stated, we turn to the conditions as we find them in cancer in man and animals, a series of analogies can be developed which indicate that in Plasmodiophora we have a parasite, the cause of a specific tumor, which, in its relation to the tissue which it infects, fulfills many of the conditions which must be required of a parasite before it can be accepted as bearing a direct etiologic relation to cancer. The investigations of Hauser, Peterson and others, have demonstrated that in primary, epithelial growths a direct continuity with the epithelium from which the tumor is derived can be readily traced. This has been accomplished by means of serial sections and wax models. These investigations have likewise shown, especially in epithelioma, a form of tumor which has been most frequently subjected to this form of examination, that the proliferation frequently begins at several points, as a rule not far distant from one another. Starting at these points the tumor spreads into the surrounding structure and through the medium of the lymph spaces, carcinomatous cells are carried into the circulation, when the phenomenon of metastasis supervenes.

Associated with the proliferation of the epithelium is, with rare exceptions, a reaction of the adjacent connective tissue. This is evidenced by the well-known round-celled infiltration at the margins of many carcinomata, by the extensive development of a connective tissue stroma and by the frequent appearance of karyokinetic figures in the nuclei of the connective tissue elements forming it. The evidences of mitosis in the stroma of the tumor are never so frequent as in the proliferating epithelium of the tumor proper. The structure of the newly formed stroma of the tumor likewise includes a more or less well-developed vascular system. That the specific factor of the tumor formation is not in the stroma is evidenced by the fact that metastatic connective tissue proliferation is not a phenomenon of the dissemination of carcinoma. On the other hand, in the regions where metastases from the primary tumor develop without regard to

the tissue or organ in which this occurs, a stroma derived from the adjacent connective tissue results from the presence of the epithelial elements of the tumor. It is thus seen that the epithelial elements carry with them and elaborate some agent capable of inducing not only the primary reaction of the connective tissue as expressed by the frequent round-celled infiltration at the margin but an extensive proliferation of the connective tissue structure which, both in the primary tumor and in the metastases, leads to the development of a more or less well-defined connective tissue framework, which forms no inconsiderable proportion of the new growth. It is generally conceded that the cells of carcinoma in a given case, are derived by proliferation from groups of cells which have primarily undergone the cancerous transformation. In most cases the cell, once so transformed, proliferates indefinitely, and transported, through the medium of the circulation, to other regions of the economy, continues its proliferative activity and in this way the metastases are formed.

It is, however, a well-known fact that every cancer cell is not capable of proliferating indefinitely. This is first shown, by the large number of cells which present high grade degenerative changes, in many cases the fore-runners of complete disintegration. It has likewise been noted that all tumor elements are not capable of producing metastases, even when transported under exactly the conditions ordinarily leading to this result. For instance, cancer cells have frequently been found in the pulmonary capillaries and in the ductus thoracicus, regions in which metastases frequently develop but which show no evidence of proliferation although present in large numbers. It will thus be seen that every cancer cell is not capable of continuing indefinitely the cancerous process.

Inasmuch as in clubroot we have no metastases, the outgrowths in this case could at best only be compared with the primary tumor in cancer. We have seen that the tumor in clubroot is composed of cells containing parasites and hypertrophic cells immediately adjoining them. The infected cells, when transplanted are capable of continuing the growth. The hypertrophic cells which in all other respects closely resemble them but which contain no parasites, are naturally incapable of producing an infection. We have seen, by analysis, that the primary growth in cancer presents a certain analogy in that it is composed of cells capable of continuing the proliferation when transported to distant regions, and other cells which morphologically closely resemble them, which are incapable of producing metastases. *For this reason we must conclude that if a parasite were the*

basis of the cancerous proliferation, it would not, necessarily be present in every epithelial cell forming the tumor. It must, necessarily, be seen that the cells which are incapable of continuing proliferation must either contain no parasites or have reached a stage in which it is itself incapable of further proliferation, in which case the organism has in all probability passed into some permanent spore-like form, a condition in which it would be incapable of reinfecting epithelium or other cells of the organism without passing through some ectogenous cycle of development. This is exactly the case in Plasmodiophora and it may be pointed out that cell inclusions in cancer which are held by certain investigators to be of parasitic nature are not present in all the cells, even when most frequent. To this must be added, however, that the inclusions in cancer which so closely resemble the amœboid form of Plasmodiophora at best represent but one form of the parasite; that at least occasional spores must be accounted for and that the organism on its entry into the epithelium is probably present in some form which, with our present methods, we are unable to recognize. The fact that the swarm spores of Plasmodiophora, when injected into the tissue, so closely approximate in their appearance certain normal tissue elements that they are unrecognizable, indicates that such a supposition is reasonable.

As the specific factor in cancer, having once developed in certain cells remains identified with them, being handed down through proliferation with certain cells of the tumor, it must greatly increase in amount. It has been suggested that this specific factor is of a toxic nature, but no explanation has been offered as to how a small amount of toxin, starting in a few cells, can continually reproduce itself. From what we know at present of toxins there is absolutely no ground for this supposition, and even in the case of enzymes or ferments, which are sometimes refered to, we have nothing analogous to the conditions in cancer. There is no doubt that the development of the stroma in cancer must be attributed to the action of a toxin, since this action is local and undoubtedly results from the presence of the cancer cells, but inasmuch as the mere proliferation of normal cells does not lead to the elaboration of a toxic substance, we must assume that this toxic substance is directly associated with the essential factor which causes the cancerous transformation of the epithelium. As the essential factor in cancer increases with the proliferation of the cells, or, in other words, reproduces itself, and we know that no toxin is capable of this, we arrive at the conclusion that the presence of an organism in the epithelium offers the best explanation of the phe-

nomena as they occur. An organism of the protozoan group, especially one more or less distantly related to the *Mycetozoa*, and therefore comparable to Plasmodiophora, would best fulfil these conditions. That such organisms, upon infecting the tissue elements of a higher animal do, in all probability, elaborate toxic substances, is shown by the facts in Plasmodiophora. A similar state of affairs has been described by Pianese, who observed atypical karyokinetic figures in the epithelial cells of the urinary tubules of the guinea-pig, which were infected by a protozoan parasite.

We have seen that in Plasmodiophora the parasite divides until the demands upon the cell are such that it likewise divides. This continues until by repeated divisions the parasite has completely filled the cell and exhausted the nourishment, whereupon the organisms are transformed into permanent spores. These spores are incapable of reinfecting the cells of the plant until they have passed through the ectogenous cycle in which they lie during the entire winter in the ground, liberating swarm spores with the advent of spring, which, entering fresh plants, lead to reappearance of the disease. The infection in Plasmodiophora destroys the growth of the plant and in a certain sense may be looked upon as a malignant infection. It is seen that the organism remains in the amœboid stage and in a state of symbiotic existence with the host cell until it is exhausted. This is dependent upon the relative rate of proliferation. It is possible to conceive that in cancer, the greater proliferative power of the epithelial cell might place it on a footing with the parasite, which would enable it to proliferate rapidly and indefinitely, in which case only exceptionally unfavorable conditions would lead to the development of spores. These spores, even if present in the tissue would be incapable of infecting uninfected cells, without passing out of the organism and through some ectogenous cycle. They would likewise be incapable of infecting the tissue cells of other animals or individuals when transplanted without the interposition of this cycle, as is the case in Plasmodiophora.

The parasite, once having entered into this symbiotic relation with the cell, would perish with the cell unless it had taken on the permanent spore formation just mentioned. For this reason it would be impossible to produce tumors, even in animals of the same type without the transplantation of both cell and parasite, which applies both to the transplantation experiments as carried on in animals, and the relations as they exist in Plasmodiophora. The few cases in which inoculation of animals with peritoneal fluid from man has been followed by epithelial

proliferation, cannot be compared directly with the conditions as they occur in nature. In the first place, while these tumors closely resemble malignant epithelial tumors as we find them in man and animals, they have in no case proved truly malignant; have never led to metastasis and have ultimately been overcome by the animal. They may fairly be compared to the endothelial nodules which develop in frogs around the implanted fragments of clubroots. The conditions in this case are entirely abnormal and the organism at best passes through but a portion of the necessary cycle. The nodules obtained in the liver of a dog by intravenous injection of peritoneal fluid from cancer of the peritoneum in man, indicate the correctness of this explanation of these rare occurrences.

In conclusion it may be fairly stated that there are inclusions in cancer which, viewed in the light of Plasmodiophora, are best explained as parasites; that a comparison of the conditions found in clubroot with those found in man, show that we are now acquainted with a parasite capable in many ways of fulfilling the conditions required of a parasite for cancer. Our efforts should, therefore, be directed to the more accurate determination of the nature and distribution of this too little known class of organisms and to the determination, if possible, of an ectogenous cycle for the parasite of cancer, which includes the possibility of an intermediary host.

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XXXVIII. SOME POINTS IN THE TREATMENT OF FUNCTIONAL DISORDERS IN THE SEXUAL ORGANS OF THE MALE

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NEW YORK

So MUCH confusion exists in the minds of medical men concerning the treatment of functional sexual derangements in the male, that I have thought a paper on this subject might perhaps prove interesting, not only to specialists in this branch, but also to the general practitioner, who himself, sees more or less of these cases, and who oftentimes is inclined either to slur their importance in a hygienic and therapeutic sense, or else, in attempting treatment, to do the worst thing that could possibly be done for them.

In order to make the subject clear at the start, let me state what the meaning of functional disorders of the sexual organs mean, so far as the purpose of this paper is concerned. They are derangements of the sexual organs which interfere with their proper functions in the matters of coition and procreation. And I shall limit what I have to say more especially to a lack of sexual and to a lack of procreative power. And first, as regards the lack of sexual power.

Let me review the principal causes which occur to produce this class of derangements, and in this class there is one which has been considered as especially responsible. The older writers, and many of the more recent ones, have been peculiarly prone to consider masturbation as the prime cause of sexual debility, and they have so taught, and the tradition has been handed down from father to son so religiously and so strongly, that it has become almost an article of faith in the minds of most doctors. My own experience in this country has been that masturbation plays a very small part in producing sexual debility, for the simple reason that American youth, and by that I mean of the male sex, masturbate comparatively infrequently, and rapidly give up the habit. They do not abuse themselves in this manner nearly as much as the youth of the continental countries do, according to the dictum of medical practitioners in those countries; but still, masturbation does play some part, although a minor one, in the production of this class of disease.

Another cause which has been heralded far and wide as a cause, is that of withdrawal; that is to say of not finishing the

copulative act in vaginam but extra vaginam, or else leaving the act uncompleted. Of these two methods the latter is far the more dangerous and the more productive of trouble, for the congestion produced by the erection and by copulation is unsatisfied and is apt to be more or less permanent and to produce more or less disturbance. The former is the less injurious because the relief obtained from the emission is much the same extra as intra vaginam, and in this form of sexual amusement as well as in that of masturbation, the danger lies more in the excitement, without the corresponding gratification, than in the act itself.

The next cause for this condition is a devotion to the opposite sex which is pursued not wisely but too well, and where the man abuses not only his own sexual functions but the gift which has been given him in the person of the opposite sex. In other words, he uses up both himself and the woman; himself, if anything, rather more, for the average woman has but little gratification in the act, is the passive agent, and can stand far more abuse in this line than the male, although we are informed by gynecologists that many of the uterine affections in the female are induced by an unsatisfied sexual desire, and this again, is produced by the male performing the act too rapidly, long before the woman has had time to enjoy the orgasm herself.

Last of all come the so-called psychic causes; that is to say mental disturbances or neurotic impressions (neurasthenia) which by distracting the attention of the male from the work in hand, prevent either complete erection or satisfactory coition in case an entrance is effected. These in their pure and simple forms, I believe are very rare, where they are not associated with some physical cause underlying the mental state. There are cases where almost complete impotence has resulted from a man allowing his mind to wander from the business, to which he should give his entire and undivided attention, by thinking of something that happened during the day, be the same of a serious or comical nature, exemplifying the old adage that a person cannot properly do two things at the same time. He cannot think well and copulate satisfactorily. Now, in a very large proportion of cases, there is some physical underlying cause for this condition and the German writers are inclined to regard gonorrhea as responsible for about ninety-five per cent. of these cases of sexual disturbances. Personally, I think their percentage is too high, for it does not follow that because there is a history of old gonorrhea in a sexual cripple, therefore gonorrhea is the cause. But I am constrained to admit that a good proportion, perhaps fifty per cent. may be accredited to gonorrheas which have remained uncured, and by uncured I mean as regards their ultimate results,

for we must recollect that gonorrhea is by no means the simple, and local disease which we have been in the past accustomed to regard it. It is a real and serious matter, fully as serious as its congener, syphilis, which has received a bad name, and perhaps not unjustly, but which, nevertheless, should not be regarded as the more serious of the two.

The physical cause is the stricture which results from an uncured clap, which is perhaps one of the commonest reasons for the existence of this condition of sexual paralysis, and while a priori, it might be regarded as almost impossible that such should be a cause, experience teaches that with the relief of the stricture and the irritation consequent upon it, the sexual disability is relieved.

Another frequent cause, perhaps running a very good second to stricture, is the presence of granulations, either with or without congestion of the urethral canal, and this also produces not only a lack of erectile power, but also a prematureness of ejaculation which robs the copulative act of its pleasure, and transforms it oftentimes into a duty to be performed, rather than a pleasure which is looked forward to.

Given then, these conditions as among the principal causes of sexual debility what has been the aim of the average practitioner as regards treatment? The name commonly given to this form of disease is sexual weakness or sexual debility; and so, on the principle of curing the disturbance by strengthening the weakened parts, he goes to work to stimulate the organs, under the impression that they are weak and require stimulation, which is about as successful as applying the whip and spur to a tired-out horse. With both the horse and the penis, the need is for rest. Sedative should be the keynote then in the treatment.

Now, how shall the sedative be applied? The majority of persons seeking relief and a very respectable number of medical men seem to think that medicine taken internally is the only thing which will cure this class of diseases. Hence all kinds of remedies are exploited, from damiana, which the advertising charlatan has claimed to have been used by King Solomon, down the entire list, including Yohimbin, the animal extracts, phosphorus, strychnia and a host of other remedies all of which are held up as the only panacea and cure-all of sexual disturbances. My experience teaches me that is a most fallacious basis upon which to treat these disturbances. Medicines, if given internally, are only useful as adjuvants, and cannot be relied upon in the treatment of this class of disease. Local applications and local remedies are the things which really do the work, and these vary from applications of the various preparations of nitrate of silver,

than which there is no better drug in our armament against this class of disturbances, to the use of sounds, hot and cold irrigations, either per urethram or per rectum, and of these the hot are generally more efficacious than the cold. But whatever the course adopted, I want to impress upon the minds of my hearers this fact; that the use of the sedative is of far greater advantage than the use of a stimulant,, and the idea that nitrate of silver and their derivatives are stimulants is a mistaken one; they act as sedatives although they may appear to operate to a certain degree as irritants at the commencement.

As regards the question of the treatment of sterility, there is, alas! but little to be said and that little amounts to an admission that little *can* be done. Probably ninety per cent. of the cases of sterility occur from an old inflammation of the epididymes, and their consequent blocking up, preventing the passage of the spermatozoa into the urethra. But inasmuch as the bulk of the semen is made up of secretions of organs independent of the testis, the emission is just as abundant as in health, but the semen is no longer fruitful. For this condition of affairs there has been practically no cure discovered, notwithstanding the ingenious operations which have been done from time to time by surgeons of various countries, looking towards the excision of the blocked-up portions of the vas deferens and the formation of a new canal which will permit the passage of spermatozoa into the urethra. A few cases have been said to have been successful, but this must be doubted on the principle that as one swallow does not make a summer, so one spermatozon does not mean fecundity.

Where, on the other hand, the sterility appears to be due either to a too thick condition of the semen or to a scarcity of spermatozoa from sexual abuse, this condition may be easily remedied, but the number of cases where this occurs is, comparatively speaking, rare and so is really not worthy of serious attention.

One other method of treatment which has been much lauded, especially for sexual impotence, either from lack of erection or from premature emission is electricity in one of three forms of Faradic, Galvanic or the Static current. So far as my own experience is a guide, I am obliged to confess disappointment in the good derived from the electrical current. Of the three, I am inclined to consider galvanism given in weak currents, and frequently administered as being the most useful. The Faradic is the worst.

The question of drink sometimesg enters largely into these cases and it is well to remember that in a sexual cripple, as in a cripple from venereal diseases, the use of stimulants should be interdicted, and that interdiction should extend not only to alco-

holic and fermented stimulants, but also to coffee the use of which, why, I do not know, seems to have a bad effect upon the sexual powers, when they are damaged in any way, and in addition, as I have already stated, the excessive use of alcoholic drinks, especially spirits is detrimental. Indeed, it may be put down as an aphorism that the chronic toper is a poor bedfellow. He is incapable in more ways than one.

In the treatment of this class of diseases, it is exceedingly difficult to impress patients with the fact that you cannot treat them at a distance merely by sending them a prescription for medicines. Of course, if we are willing to descend to the methods of the advertising charlatan, we can humbug people to the utmost, dose them with medicine and leave them at the best no worse off than they were before. And I sometimes wonder, my brother doctors, when I regard the multitude of fools who fuss and potter over their sexual organs and their sexual conditions, whether it is worth while to try and prevent them from humbugging themselves. The caustic old Roman was not far wrong when he said: "Populus vult decipi; decipiatur ergo," for people really love to be humbugged provided it is done properly and not too baldly, as I think the high priest of humbug himself admitted when, having retired from business he described the manner in which he wheedled the tardy dollar from the pockets of an easily gulled public.

DISCUSSION

Dr. R. H. M. DAWBARN, of New York: There are two operations, looking at this matter from the surgical standpoint, which are of some benefit in aiding those who are jaded in their sexual powers and who are unable to maintain an erection or have the orgasm too quickly. I am one of those who believe that Moses knew his business, and that we as a profession should consistently advocate circumcision. I do not refer to its great value in the prevention of syphilis and chancroids. I have repeatedly performed the operation on adults, and the invariable report from them has been that they enjoyed intercourse much more than before the openration, because the orgasm did not come so quickly. A second operation which should be thought of is for those who can not keep up the orgasm sufficiently long, owing to varicocele of the veins of the penis itself. The tying off of a few of the veins at the base of the organ make a wonderful difference in the vigor and permanence of the erection. However, the operator should be extremely careful not to tie off one of the accompanying nerves. The first time I did the operation I made this mistake, and the patient haunted me, telling me that although the erection was better the sensibility of the organ had been reduced by my tying off the nerve.

There are a number of instances which tend to a different conclusion from that of the author of the paper with regard to the effect of operations on the vas deferens. For instance, Dr. A. G. Gerster has reported a case

in which the vas deférens was accidentally divided during an operation for hernia. The operator took a piece of the smallest catgut obtainable, one centimeter long, introduced it into the two ends of the vas, and brought the divided portions of the vas together over it with four fine interrupted silk sutures. He examined the patient two years afterward, and the testis was firm and apparently normal on that side, and the patient said that there had been no discomfort following the operation. This is an important point, because Lombrosos has recently advocated division of the vas, whereas formerly he advocated castration in habitual criminals. A knowing criminal might, therefore, subsequently have the vas deferens resected and its function restored.

Dr. A. JACOBI, of New York: What has been said by the last speaker concerning circumcision appears to me to be worthy of consideration, not from a surgical, but from a general medical standpoint. If it has any effect I believe it is more the effect of suggestion or hypnotism than any other, for, there is not a better way of curing impotence than to act upon the nervous system.

This leads me to say a few words upon the subject of masturbation, which has been touched upon in the paper. Masturbation is, after all, a very powerful cause of impotence, not that it really weakens the generative organs very much, but because it weakens the self-consciousness and the courage of the person who has suffered from it. Masturbation, when continued a number of years, will, in the great majority of cases, result in frequent nocturnal emissions, and the latter weaken these persons very much more than the maturbation. The self-confidence of the person is impaired thereby, and it explains why so many newly-married men can not cohabit. They should be told positively that within a few months they will be better. If these persons once succeed in cohabiting properly they will be cured; if not, it will take some months more before the requisite self-confidence is attained. I, therefore, believe that circumcision will do good in a number of cases; it impresses the unfortunate person with the belief that something of importance has been done for him.

Dr. STURGIS: My paper has brought out a discussion, and I am very glad to answer the speakers. How will Dr. Dawbarn explain the fact that many cases of impotence and sexual debility occur in those who are already circumcised? This operation is by no means a cure-all, although it may be occasionally of benefit. Circumcision, I do not believe, is useful except where there is a very long and adherent and dirty foreskin. The tying of the veins I would place in the same category. The mere fact that there is a large seminal emission does not necessarily mean that there is procreative power, because the semen is made up largely of the secretion of organs entirely independent of the secretion of spermatozoa. Martin, of Philadelphia, has performed several operations of this kind, and the majority of his cases showed no spermatozoa present, although the seminal emissions were large. The obstruction usually persisted. In one or two cases, although immediately after the operation a few spermatozoa were present, they were absent altogether later on.

With regard to Dr. Jacobi's remarks, I also believe that the operation of circumcision produces a nerve impression, yet I believe this nerve influ-

ence is not important except in those who are distinctly neurasthenic in sexual matters. The nerve influence, I believe, is exerted through a physical cause, just as alienists believe that insanity has a physical basis.

With regard to the weakening effect of masturbation, I wish to say that in this country I have been struck with the fact that ninety-nine per cent. of men masturbate, but they feel ashamed of the habit and conceal it. It does not have a weakening effect provided it is not done too frequently; the effect is no more debilitating than from proper sexual intercourse not carried to excess. I have found that many widowers masturbate, but only just enough to relieve their sexual desire. I am not in any way an apologist for masturbators, but I am speaking to doctors about a matter in which I think I have had a fairly large experience.

XXXIX. THE TECHNIQUE OF PROSTATECTOMY.

By RAMON GUITERAS, M. D.

NEW YORK

In closing my remarks and my address before the Section of Urinary Surgery at the Thirteenth International Medical Congress in Paris, in August, 1900, I said that "I considered the operation of prostatectomy still in its infancy in the same position that hysterectomy was twenty years ago; but that I had no doubt that it would some day be simplified, and that, therefore, it behooved all surgeons interested in this line of work to improve the methods and technique of the operation." When making these remarks I little thought that prostatic surgery would advance with such rapid strides as it has, and that the operation would be of so great interest to all surgeons. That such is the fact, however, may be easily discovered by reading over the surgical literature of the last few years in which it can be noted how many surgeons have written upon this subject, and how many and varied have been the techniques employed by them.

In other countries the urologists have been foremost in perfecting this line of work, and the transactions of the last two years of the French Association of urologists have been rich in the description of new and scientific methods of performing this operation, which but a short time ago seemed to baffle the ingenuity of all operators. In this country, notwithstanding the fact that much good work has been done by the urologists, the general surgeons have, perhaps, been the most active in pushing this line of surgery, so that to-day in almost any community an operator can be found who is able to remove a prostate successfully. In fact, only a few nights ago in a discussion upon this subject in the New York Academy of Medicine, one of our ablest

surgeons said, that he considered prostatectomy a very simple and easy operation. The reason why it is now considered more simple is not only on account of the improvement in the technique, but because operators are better able to judge which prostates they can remove and which they had better leave alone.

The indications for prostatectomy are repeated attacks of acute retention of urine, urethral fever, epididymitis, or hematuria, in a patient whose general health is good and whose kid-

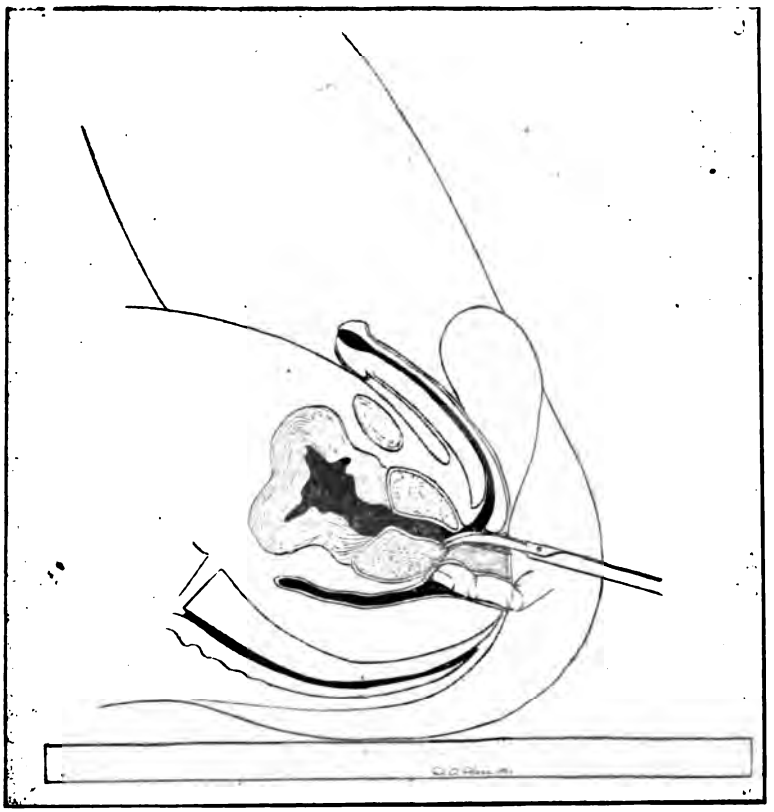

Fig. 1.—Cutting through floor of prostatic urethra with curved scissors.

neys are sound. The contra indications, on the other hand, are extreme old age, general debility; valvular heart disease, or fatty heart; marked arterial sclerosis; medical or surgical disease of the kidney; or the existence of any other chronic trouble which would, perhaps debilitate the patient to such a degree as to render him unable to endure an operation. Regarding the

condition of the bladder, it may be said that it is less important than that of the kidney, for it makes no difference how badly involved the bladder may be, it will probably be benefited by the use of internal antiseptics and irrigations once the obstruction has been removed. It is surprising to see how often the bladder can empty itself almost entirely after a prostatectomy, even if it has been previously considered atonic, and years have been passed in catheter life. The shape and consistence of the

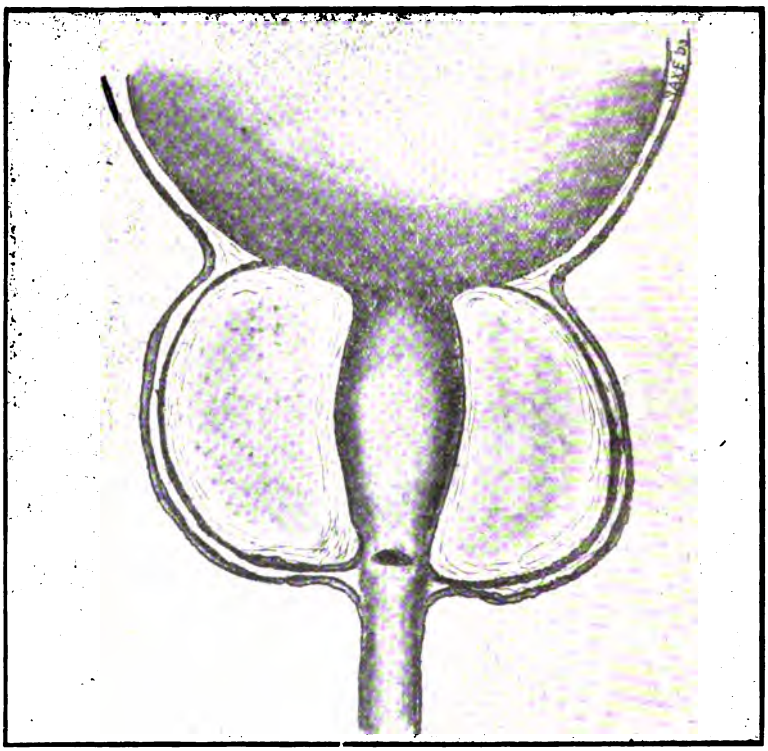

Fig. 2.—Showing opening in floor of prostatic urethra made by scissors through which finger is introduced in enucleating gland.

prostate must also be considered, as enucleation is indicated in the large -sized prostate, and not in the small sclerosed glands, or collar-shaped contractions that give rise to urinary obstruction.

All cases then in which prostatectomy is not indicated should either be operated upon by the Bottini method, permanent suprapubic drainage, or else relegated to catheter life. The following table will indicate roughly the factors governing the choice of treatment in prostatics:

FACTORS GOVERNING CHOICE OF TREATMENT IN PROSTATICS

	Indications.	*Prostatectomey.*	*Prostatotomy.*	*Catheter Life.*
1.	Age....	Under 70 years.	Any age over 50 years.	Any age over 50 years.
2.	Kidneys..	Normal.	Fairly healthy.	Considerably diseased
3.	Bladder.	Fairly healthy.	Fairly healthy.	Atonic.
4.	Prostate.	Markedly enlarged as felt by rectum.	Moderately enlarged as felt by rectum.	Any size.
5.	Urethra.....	Decided impediment. Elongation of the canal.	Distinct impediment.	Catheterization possible, without difficulty or complications.
6.	Residual urine.....	Over four ounces.	Over four ounces.	Over four ounces.
7.	Special symptoms.	Frequency of urination, pain, tenesmus and burning.	Frequency of urination, pain, tenesmus and burning.	Frequency of urination, pain, tenesmus and burning. Heart feeble, arteries diseased.
8.	Complications.....	Attacks of urethral fever, apididymitis, and hematuria (kidneys normal).	Same, but kidneys only fairly healthy.	Same, but kidneys too much diseased for operative proceedure.

Prostatectomy was practically introduced into surgery by Bellefield and Magill, in 1886 and 1887. Magill first began to enucleate obstructing portions of the gland through a suprapubic incision into the bladder and gradually increased the amount enucleated until the entire gland was shelled out. Bellefield pursued the same tactics, but added a Bouttoniere perineal urethrotomy to the operation for the sake of draining the bladder from below after the suprapubic prostatectomy. Fuller then followed, but assisted the enucleation through the bladder by pressure with the fist on the perineum below. The author pursued the same tactics excepting that he made counter pressure with two fingers upon the gland through the rectum.

Suprapubic prostatectomy is, however, I believe, gradually becoming less popular and is being supplanted by the perineal route. Van Dittel was one of the first to remove a prostate in this way by a coccygeal perineal incision. Then came Zuckerhandl and Pyle with semilunar and transverse incisions in front of the rectum. The French surgeons have almost unanimously adopted the method of approach by the transverse incision in front of the anus, dissecting back the rectum, cutting through the levator ani muscle and the external capsule of the prostate, and removing the gland through this incision. Wishard, in 1892, removed the gland through a intraurethral incision; Nicholl, in 1895, performed a perineal enucleation, making pres-

sure from above through the bladder. Alexander followed, performing an enucleation through the perineum by means of an intra urethral incision, cutting through the capsule of the prostate, also resorting to a cystotomy for pressure on the gland from above. Shortly afterwards, in 1899, Bryson performed an enucleation through the urethra, resorting to suprapubic pressure from above in Ritzius space without opening the bladder. Syms, the same year, performed a perineal enucleation through the urethra, resorting to pressure from above through a suprapubic opening into the peritoneal cavity. In 1901, however, he again

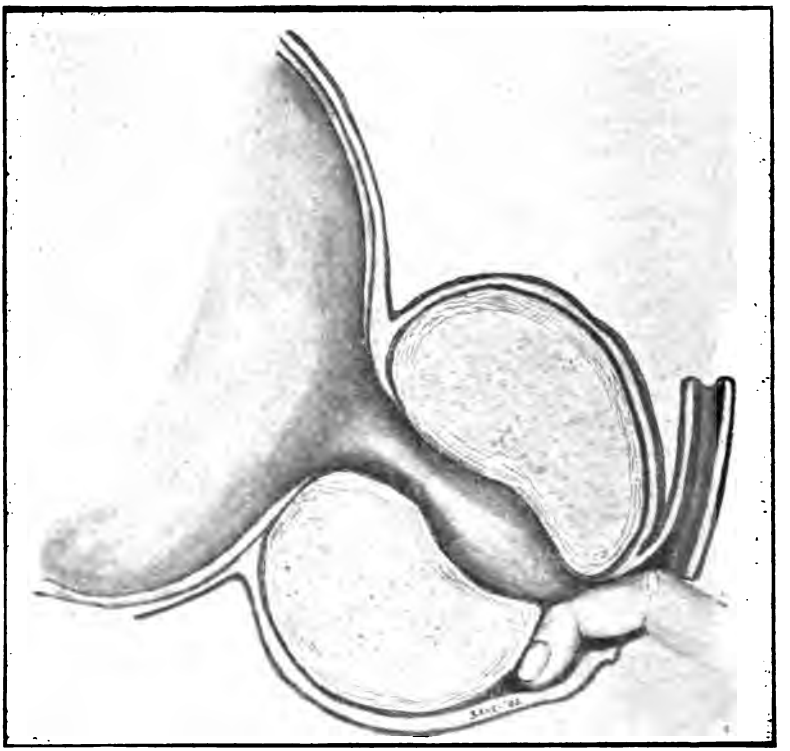

Fig. 3.—Finger passed through opening in Fig. 2 and enucleating between capsula propria and external capsule.

modified this operation, using a balloon passed into the bladder through the perineal incision in a collapsed state, which he then inflated. By this means he was able by exerting traction upon the balloon to force down the gland during its removal.

During the time spent working in this interesting field I found, that, although in some cases prostates could be shelled out

through the perineum without pressure from above on the base of the gland either in the bladder or Ritzius space by simply hooking the end of the finger over the gland in the rectum, that the perineal operation would be materially aided by practising traction on the base of the gland. I accordingly investigated the merits of Syms' rubber balloon, and was so much pleased with the device that I presented it in delivering an address at the Thirteenth International Medical Congress in Paris, in 1900. Later on I fancied that the traction by this method was not firm enough, and could not be applied to the exact locality in which the enucleation was going on. I accordingly had a pair of forceps made resembling sponge holders, but with oval ends to the blades, the inner surfaces of which were striated so as to prevent the gland from slipping after it had been grasped. (Fig. 6).

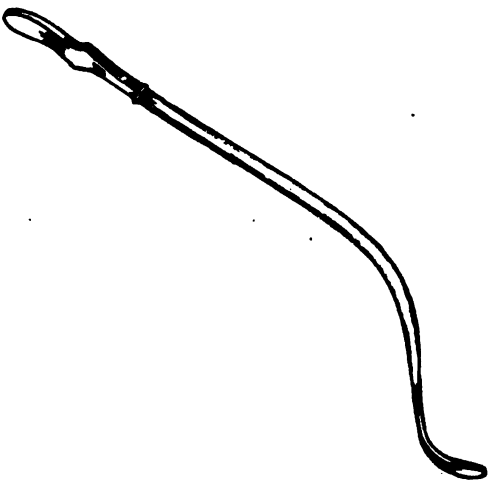

Fig. 4.—Prostatic Depressor (Guiteras). For prying down the prostate during enucleation.

There were, however, certain difficulties encountered in applying these forceps, as it was difficult to steady the lobe of the gland while they were being applied. I was, therefore, led to use a depressor that could be passed through the perineal incision into the bladder and, having hooked it behind the prostate, I could by exerting traction pry the gland down while enucleation was being performed. This instrument was shaped somewhat like a stove-cover lifter, and could be turned in almost any direction in the bladder, thus exerting pressure on the part of the gland where it was most desirable (Fig. 4.) Similar instruments have been advocated by Gouley, of New York, and Ferguson, of

Chicago. The prostatic depressor used by me, and which I now present, is in reality a sound bent into the desired shape with the end flattened. A similar contrivance has been made from a vaginal depressor bent in the same curve. The advantages of these instruments is to steady and force down the gland while it is being removed. These instruments illustrate in prostatic

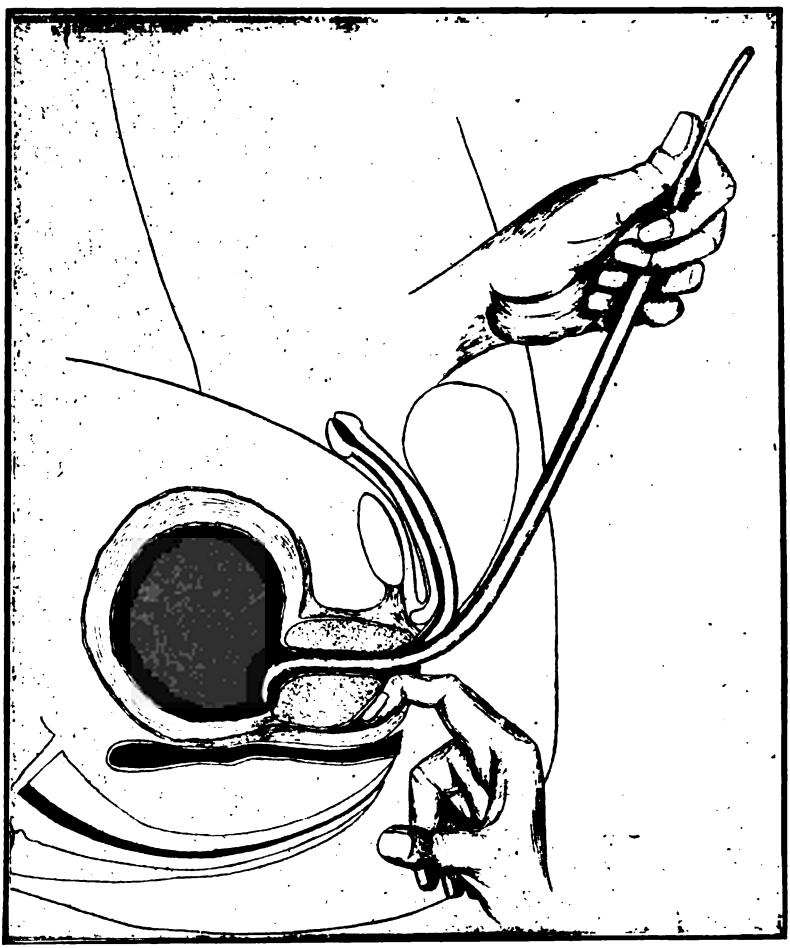

Fig. 5.—Showing depressor in position.

surgery the old and popular adage, "pull, and if you can't pull, push."

The technique of the operation of prostatectomy as advocated by me at present is then as follows: The patient should be pre-

pared as usual, the perineum and suprapubic regions should be shaved, and the rectum and bladder washed out clean before the operation. He should then be etherized and placed upon his back upon the operating table. A lithotomy guide is then passed through the urethra into the bladder and the patient placed in the lithotomy position. An external perineal urethrotomy is then performed, opening the membraneous urethra, after which the forefinger should be pushed through the perineal opening into the prostatic urethra with the object of examining it, and as much of the bladder as possible by palpation. This opening should then be dilated with the fingers, or instruments to a sufficient degree to introduce the blades of the scissors for cutting through the floor of the prostatic urethra.

With the fingers of the left hand in the rectum, pressing

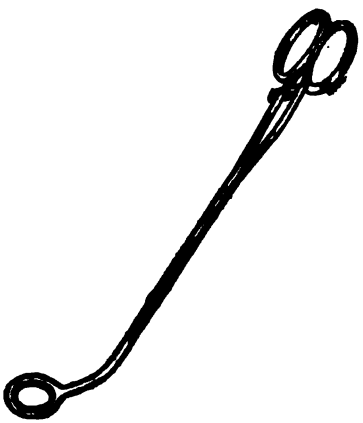

Fig. 6.—Prostatatic Forceps (Guiteras).
For removing lobe of gland after freeing it from external capsule.

upon the gland, I introduce the point of the blades of a curved pair of scissors into the urethra until they have just passed the apex of the gland, when I make a transverse incision in its floor. The tip of the forefinger of the right hand is then introduced through the urethral incision and gradually works its way between the gland and the external capsule, separating it from this latter covering.

The depressor is then introduced. This, owing to its curve, can be held in the upper part of the perineal opening entirely out of the way of the finger and does not interfere with it at all. (Fig 5). When one lateral lobe is freed the forceps are placed upon it, and it is delivered. At times the base of the glands cannot be easily freed, in which case if the forceps are put on and

traction made, the finger can break up the adhesions and the lobe can be gently withdrawn. The prostate gland usually comes out in two pieces with a so-called middle lobe adherent to one of the lateral ones; but it sometimes remains behind after the two lateral ones have been removed, in which case it can usually be loosened with the finger-nails, or grasped and brought out by the forceps.

The gland having been removed it is important to apply a pair of artery forceps to either side of the upper margin of the urethral incision and then sweep the finger around between the

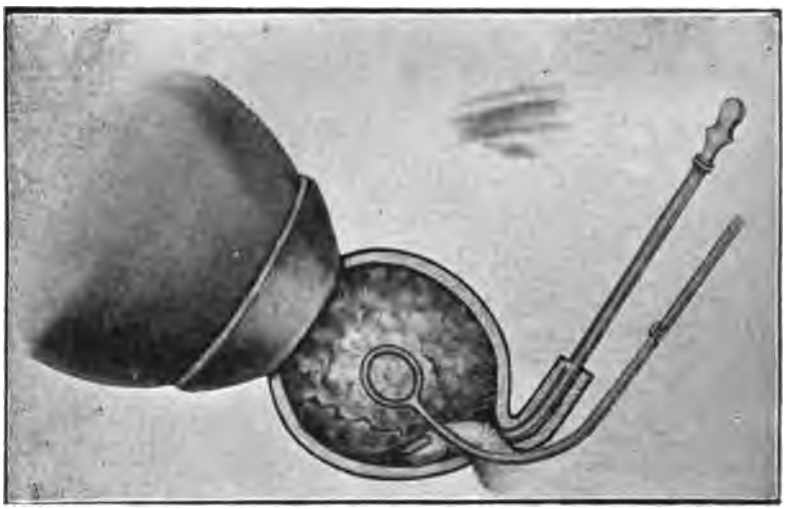

Fig. 7.—Showing traction-forceps on a lobe of the gland. Also the depressor passing through urethra into bladder. The right half of external capsule has been removed by vertical incision. The collar on the neck of bladder is the reflection of same part of fascia which has been cut off at right angles to prostatic portion.

urethra and the capsule, and introduce it through the urethra into the bladder to see that everything is free. The bladder should then be flushed out with hot water, and if any calculi are present they should be removed. A second irrigation of 1 in 10,000 bichloride solution is then given, and a drainage tube inserted into the bladder through the perineal incision. It must be remembered that the artery forceps just referred to should be allowed to remain on the upper edge of the incision until the perineal drainage tube has been introduced into the bladder, as it often is extremely difficult to make the tube pass through the urethra if its floor is not under control; besides

which there is danger of tearing the canal, or introducing it between the urethra and the external capsule. It occasionally happens after performing a very rapid perineal prostatectomy that it requires more time to introduce the drainage tube than it had for the operation. A large gorget passed in over the ends of the forceps facilitates the introduction of the tube. The skin of the perineum is then closed tightly up to the tube and the operation is completed. The tube remains in the same position that it does after an ordinary external urethrotomy. It is removed at the end of a week, after which a large-sized catheter is passed through the entire urethra into the bladder and allowed to remain until the urethra closes about it and the perineal incision has filled in. The care of the bladder is the same as in any other condition in which a retained catheter adeneum is left in situ for the same length of time.

XL. THE SURGEON'S ENEMY, THE SKIN.

By ROBERT H. M. DAWBARN, M. D.

Professor of Surgery, New York Polyclinic Medical School; Surgeon to the City Hospital, New York.

It is the chief purpose of this paper to offer a suggestion which is perhaps original, and upon which the writer has been experimenting for several years past. This suggestion, which is extremely simple, has to do with our continual fight against wound infection from microbic activity, especially in the skin.

The skin is no friend to the surgeon. It is admitted now that sterilizing it is impossible, though we may in practice somewhat approach this condition in (a) instances where the surgeon or patient chances to be one of the fortunates—in this regard—who rarely if ever sweat visibly; or (b) in those operations which do not demand a high temperature of the air in the operating room.

As to the former, (a), without question the difference in vital statistics involving percentages of aseptic results as between equally well-trained operators, mainly depends upon this factor; the "personal equation" depending more upon activity or inactivity of sweat and sebaceous glands by the million than upon any other one cause. Of course, this statement refers to operators who still adhere to naked-hand work.

As to the latter, (b), whenever an operation involving opening the peritoneal cavity is to be performed—and this includes nowadays invariably herniotomy—the careful operator avoids one cause of shock, namely, from chilling the bowels and the abdominal sympathetic, by operating in a room the heat of which is as nearly as can be that normal within the belly. Hence as a rule the patient's skin and that of most operators will be sweating, and if the spermatic cord—or a loop of bowel—be turned out even for a moment in contact with this skin, then even though irrigation be used, the prospects of aseptic healing are not bright. This is so true that some of the ablest operators in New York, in incisions in the uro-genital area, and particularly in their hernial work, do not try to close up the skin wound upon the day of operation.

The sweat and the sebaceous secretion are never sterile. This fact deserves italics. Though at the moment of beginning an operation the patient's skin has had the usual twenty-four hours' preparation, winding up with a final scrubbing with soap, alcohol and finally ether, yet the instant this skin becomes dewed with

beads of sweat, and in some regions shiny, too, with oil, it is a threat, a menace; and one which constitutes the surgeon's chief enemy, so far as aseptic results are concerned. Our instruments, dressings, ligatures and sutures, caps, gowns, gloves, can readily be sterilized by heat. In emergency a pot of water boiling on the kitchen stove is alone needed to secure for all these essentials and accessories of operation freedom from living germs, and to produce results just as good as those obtainable by use of the most expensive steam sterilizers. But as to the sweating skin, the problem is much more difficult.

Plainly, it is a desirable thing for both operator and patient to use very hot water, as hot as bearable, in preparatory cleansing, to promote activity of the skin emunctories in question, before operation, so that there shall be a flushing out by means of the running sweat. But even so, the purest of sweat is impure; it is never sterile; and the sebum is even less so.

The risk to the wound from contact with the naked skin of the operator, due to his secretions, or from the patient's own skin, is so great that if we are to go ungloved, we may well give hearty assent to the views of Kœnig, who at the Medical Society of Berlin (December 10, 1902) discussed the subject of how best to avoid such danger. His suggestion entails the absolute avoidance of even the smallest contact between the surgeon's skin and the wound depths: and to this end he has had made special instruments with very long handles, so that even in deep wounds the fingers can be kept out. He states that in his last thousand operations, he has performed six hundred by this method, of which one hundred were joint and bone operations; and in none of these six hundred was there the least disturbance of asepsis following the operation.

As a point of some interest we may mention that certain operators habitually take up a fresh knife after section of the skin, lest microbes clinging to its blade, and coming from the lower layers of the true derma, may thus be conveyed into the deeper wound. Certainly a momentary vigorous wiping of the scalpel thus soiled is at least commendable.

As a teacher of surgical technique, the author of this paper finds that the tendency of the occasional operator—the general practitioner—to finger the wound he is making, where instruments (ordinary ones) would do as well or better, is a most noticeable one, and is one of the first mistakes which he should unlearn.

The writer believes that it is the duty of the surgeon, after devoting all possible care to the cleansing, both mechanical and chemical, of the skin of both patient and operator (of which more later

on), to cover every exposed inch of such prepared skin with material that has been boiled or baked. This being so, the custom so prevalent of ending the sleeves of the operating gown at or above the elbows is illogical, and consequently, he thinks, indefensible. How often are the elbows of operator and assistants as carefully scrubbed and then sterilized chemically as are the hands? And yet those elbows all through the operation rub against the towels, gauze sponges, instruments, etc., collected about the wound. The sleeves should be as narrow as will permit the hand to pass through; and at the wrist a broad elastic is basted in, just comfortably fitting when relaxed. This, if of good rubber, will stand boiling with the gown quite a number of times, and is much less bothersome than strings or tapes. The gloves go high enough to cover the sleeve ends; thus leaving no naked skin.*

The writer believes, with a majority of the surgeons of New York to-day, that *rubber gloves have come to stay*. Steadily they have won their way, year by year, until now it behooves the minority to defend its position if it can. And not alone are they the means of effecting a greater percentage of aseptic results; but also they should be used in all infected cases. For years past, the writer can hardly remember an operative case in which he has not used them: in asepsis, to protect the patient; in other conditions, to protect the surgeon.

The statistics published in the Philadelphia *Medical Times*, by Dr. Prince A. Morrow, showing the enormous percentage of syphilis in the public hospitals of France† must cause one, reflecting on the fact that poor human nature is not widely different the world over, to feel more comfortable while operating with moderately thick rubber gloves upon his hands—with their numerous small abrasions for ready entrance of poison—when he does not know the sexual life-history of his patient. Who among us does not know a number of prominent surgeons the victims of syphilis acquired in the course of their professional work—the chancre appearing upon the hand? And the writer has had the dread experience of losing by death from syphilis of the brain a friend who, acting as house surgeon in a New York hos-

*It is the writer's custom in operating at the City Hospital to have always present some experienced member of the resident staff to act as censor of asepsis. He is to watch for breaks, and instantly to call attention when need arises. If the person at fault is the operator, so much the more hearty are the thanks accorded the censor. The theory seems to obtain that the assistants are generally the defective links in the chain, and that the surgeon is above suspicion. On the contrary, the latter seems to me the man with most reason to scratch his head.

†During the past one hundred and one years, France has enforced a certain form of regulation of that vice which lends its name to all the others—to invert Du Maurier's phrase—and such regulation has at least helped the study of the subject.

pital, pricked his finger while sewing a cut on the lip of a stranger who happened in. Casually this man remarked before leaving that he was taking blood-medicine: and upon examination this proved to be mercury. Promptly on time came the chancre where the needle had entered the finger; and though the regular course of treatment was followed, some years later when supposed to be cured he suddenly lost his memory, and went from bad to worse and to death from gummata of the brain, as stated. How many times has each of us who are surgeons risked through a needle-prick or other similar accident, while operating, a like ending to our lives? Certainly dozens—and only by an overruling Providence have we been spared what others have undergone. But (and here is the point) if that poisonous needle had first been wiped off by passing through a moderately thick rubber glove, certainly the chances of syphilitic infection would have been greatly lessened. The same is true as to the risk of pyæmic infection during operation upon septic cases.

The folowing are the objections to use of rubber gloves ordinarily heard among those who still abide in outer darkness:

1. They are an excuse for dirty surgery and unclean hands.
2. They are a source of heavy expense.
3. They are slippery in handling tools.
4. They interfere with the sense of touch.
5. When torn or cut during operation they allow sweat (which by heat they invite) to pour forth into the wound.

As to the first objection, surely now that a majority of well known surgeons are using them no reply is needed. Regarding the second, this is more true or less, according to the inexperience, or the reverse, of the wearer. I think, too, that there is a noticeable difference herein between *meum* and *tuum*. When a man is paying for the gloves, somehow he does not tear so many as he used to do in the days when he was a hospital interne. Perhaps this difference is wholly due to increasing experience; perhaps, again, he finds, upon beginning private practice, more time to draw the gloves on and off with care.

By doubling the wrist (gauntlet) of the glove, and pulling upon it so doubled, one does away with the chief source of tearing; and proper lubrication avoids the need of ever pulling at the individual fingers. In the instance of gloves but a few times worn or boiled, if of good make and proper size, tearing is infallible evidence of inexperience.

A few practical points just here may not be amiss. We should avoid cheap gloves. They look all right at first, but do not stand wear and tear. One is reminded here of the Chinaman's bit of

profound philosophy in Pidgin-English: "Cheap things no good; good things no cheap." Again, it pays best to procure thick gloves. At first I reversed this rule. When by testing with the æsthesiometer I proved how little my sense of touch was spoiled by the thickest ordinary make, and noticed how much better these lasted, and finally reflected that a poisoned needle would be cleaned off just in proportion to that thickness—I changed my programme.

Again: one should buy his ordinary kid glove size. It is equally a nuisance to wear gloves too loose, and hence with wrinkled fingers, and to use those tight enough to benumb by elastic pressure the sense of touch. Boiling results in a slight increase in size—the gloves grow looser; but this, of course, is less noticeable in the thicker ones. It is well to keep under water any gloves not shortly to be used. To avoid this becoming slimy, use for instance a saturated solution of boric acid. One of the largest dealers in rubber goods of all sorts told the writer years ago that stock which he did not expect soon to sell he always kept under water. Exposure to air or contact with oil of any kind will "kill" rubber quickly. The operator should remember that it is ruinous to rubber gloves to handle any sort of surgical adhesive plaster; ditto as to collodion, so often used (the flexile) in sealing wounds after suturing.

The mending of torn gloves is an art worth cultivating. For tears of the fingers, it is possible to patch or put on a new finger taken from a glove hopelessly spoiled, as follows: Get several sizes of thick urinary test tubes—thumb calibre, medium and small. These are to be used as a housewife uses a wooden egg in darning stockings. Melt the surface of the patch and glove finger just about the hole—or of the overlapping region if a new finger is in question—either by aid of the Paquelin cautery or of the practically identical device now sold for $5.00 (five dollars), and employed in making artistic burnt work upon wood and leather. Apply the patch and bind it very firmly in place against the glass tube within, by aid of strong, soft twine; leaving this on for a half day. I have found this plan to result in a glove which will even stand boiling again. But in the matter of patches it is best to turn inside out the glove about to be worn—so that they will be inside the finger; this just for entire safety against their dropping off and becoming lost in the wound.

A quicker and less troublesome way of mending is the following, but it does not stand boiling: First, roughen both patch and glove by aid of finest emery paper; then apply freely good bicycle rubber cement; and when this is almost dry, apply and bind on the patch, just as before described.

Still simpler and even quicker, but clumsy in appearance, and like the forgoing not tolerating boiling, is the use of surgeon's rubber plaster for patching; either the old fashioned kind requiring heating to adhere, or that now more commonly at hand —the zinc oxide plaster.

Such mended gloves I keep in any mild antiseptic solution, for instance a one per cent. lysol, and use them for pus work. The lysol solution makes a good lubricant, and attacks too any microbes in the sweat of the hands. Stronger than this percentage is irritating to most skins.

To sterilize good rubber gloves, the following is the plan used in the hospitals with which the writer is connected. As many pairs as needed are first filled with hot water, wrapped in a towel, and boiled for just five minutes, no longer; and a weight of some kind is used to keep the package, which inevitably contains some air, from floating to the surface meanwhile.* Then with sterile hands and gauze the nurse dries them while hot, using a well-known trick, better seen than described, to use air-pressure instead of finger-pulling in turning each glove inside out for this purpose. Next, they are dusted thoroughly, inside and out, with my special powder, to be described presently, and the gauntlets are folded back two inches or so—ready to pull on; and finally they are placed, each pair neatly folded and pinned, in a sterile towel, which has a label fastened upon it, "No. 8" or whatever size they may be.

Regarding objection No. 3, gloves may now be obtained the surfaces of which are not in the least slippery, even when wet; and of course these are preferable to the smoother ones.

As to No. 4, I find when tested with the æsthesiometer, my own sense of touch is much less impaired, even with thick gloves if they fit properly, than I had anticipated—not over ten per cent. —and for by far the greatest part of our operative work in wounds, this is a negliglible quantity. The instances where a very delicate touch is necessary may be met by drawing off the gloves, feeling with the naked fingers, and then replacing them.

The writer considers objection No. 5 the only one worthy of the enemy. Indeed, when gloves are lubricated by the methods in common use, it is unanswerable. Quite recently, and significant of this fact, so eminent a surgeon as Dr. George Ryerson Fowler, of Brooklyn, New York City, published an article in which, although formerly a friend of rubber gloves and regularly for years employing them, he expressed his feeling that the ad-

*Thus the nurse tests for possible needle holes. All gloves leaking even a drop must be mended before use. Carelessness at this point is inexcusable; and the surgeon to protect his patient against it should test with air for an instant each glove before drawing it on.

vantages are outweighted by this great disadvantage; namely, that they promote sweat—never sterile—and when torn during operation, an accident always liable to occur, this infective material instantly pours out into the wound.

It seems to the writer that there is but one effective reply to this objection to rubber gloves, otherwise a fatal arraignment of them. This reply is the suggestion which is the chief purpose of this paper, and one which, although very simple, is none the less a gain in surgical technique—namely, the employment as a lubricant of a powder at once efficient for that purpose and also both a vigorous astringent and an antiseptic. The search for the best means of combining these three desiderata has not been hastily conducted. For several years past, with the objection in question as a cause, the writer has both at the City Hospital, New York, and the Polyclinic Hospital tried a variety of powders. It would not be profitable to discuss those tested and dropped for various reasons. The one used for more than a year past, and which works well in practice, is a combination of one-half lycopodium and one-half official alum. These are well mixed, carefully corked and the cork tied in, and then the mixture fractionally sterilized in the steam sterilizer at the highest obtainable temperature for at least an hour, upon three successive days. Rubbed freely and well upon the hands and used within the gloves, this powder renders the hands entirely dry during operation. The tendency to sweat is at once checked. And if, during an operation, a glove should be torn, not sweat but at the worst a little of this sterile powder would escape. This, bright yellow in color, is readily seen and removed; though were a few particles of it to remain by oversight, it would hardly be liable to cause trouble. At once, the tear having occurred, either the glove is to be changed or else a sterile rubber finger-cot (of which a supply should always be ready) is drawn on over the torn glove finger.

There are two causes of tearing gloves which are so common and yet so readily preventable as to require a brief comment. The first is the use of sharp retractors, a practice all too common, and resulting in a series of punctured wounds, not through the skin, but in the deeper operative field; and such punctures can but add to the traumatism and the following day's discomfort. The operator should in a large majority of instances not use such an implement; instead, blunt retractors, and indeed, often the use of dry aseptic gauze or the edge of a dry sterile towel will serve equally well, and at the same time avoid the risk to the gloves.

Again, a surgical needle ought never to be drawn forth with

the fingers alone, whether naked or gloved. It is inviting trouble. If the surgeon prefers not to use a needle-holder—and it is admitted that except for small needles the best holder is the one the Lord made—then let the operator seize its point, in order to withdraw, through the medium of the ordinary gauze sponge.

As an item of economy the use of closely woven cotton or lisle-thread gloves is in certain ways to be encouraged; that is, upon the hands of nurses or assistants who have only to handle dry materials; but he would deprecate their use in the wound-depths, to replace rubber gloves. It has been repeatedly demonstrated that they do not prevent to a degree at all effective the passage of microbes, when wet;* not protecting then either patient or surgeon.

Regarding the control of the sweating skin about the patient's wound, three several principles now in use by various surgeons are to be noted:—First; simply the absorption of such sweat, as it reaches the surface, by the use of any sterile bibulous material placed upon the skin about the wound. Second, a sealing-up of the mouths of the sweat glands and sebaceous glands by means of a plaster specially prepared for that purpose. Third, the author's plan of checking the activity of the glandular system of the skin about the region of the wound, by free application of the astringent powder already discussed, rubbed in and left in abundance under the wet, sterile dressings for an hour to two hours before operation; the time varying according to the region of body and the sweating proclivities of the patient; also according to whether the operating room is to be at a high temperature or not.

As to the first of these principles, it is carried by some laparotomists, anxious because realizing the risk from the skin, to extremes; which are however logical ones. Such, for example, is the plan regularly used by certain surgeons, of beginning by fastening a large sterile towel in place with its middle over the proposed wound. The first cut is through the towel, the second through the skin; and now the operator stops long enough to whip together rapidly, by a running temporary suture, these cut edges of towel and wound. Thus he makes certain that nothing turned out of the wound (the spermatic cord, for example) can touch even momentarily the skin surface. Further, this running suture

*Since this paper was read Professor Mickulicz has held public clinics in two of our New York Hospitals In both instances he used cotton gloves, and as fast as wet with blood removed them and drew on a fresh pair. More than a dozen pairs were used by him in this way during a not long clinic. I think few surgeons will be inclined to change to this programme, although it is certainly an improvement upon the naked hands, since the cotton gloves absorb sweat. For several years past the writer has made use of these gloves for dry work, as previously stated.

is protected from doing damage to asepsis, by passing its needle always from below upward; thus avoiding dragging microbes from the deeper layers of the cutis vera downwards into the subjacent tissues; and between each two stitches for a like reason the suture is carefully if quickly wiped off with a wet antiseptic sponge.

Most operators however simply arrange towels about the wound region as closely as possible, and take every precaution to leave no unnecessary exposure of surface.

The second plan is credited to Dr. Murphy, of Chicago. It is a thin, sterile sheet of rubber, the surface of which upon the side toward the skin, is covered with adhesive material so tenaceous as to block effectively the mouths of the glands. This is first applied, and then the wound is made through its centre. Also, in concluding, the skin-stitches are inserted through it; and finally it is stripped and cut away from amid such stitches and the surrounding surface. About $2.00 is the cost of a sheet so prepared, of amply large size for a major operation.

As to the third plan, the writer's thought has been that here, as elsewhere, prevention is better than cure, the prevention of secretion rather than trying either to absorb it from the surface or to dam it back.* The method has the advantage of simplicity and economy; it is effectual in procuring a dry skin, though elsewhere the patient may be sweating freely; of this anyone can readily convince himself. Rubbed in by the aid of a sterile gauze sponge, and with a few extra drachms left beneath the loosened dressings, a couple of hours later it is flushed away during the final scrubbing-up of the operative field. But now the surgeon may feel easier in his mind; for his enemy, the skin, is no longer capable during operation of jeopardizing the asepsis for which he is striving.

*It is understood that it is chiefly in laparotomy-work, including in the term of course all hernias, that the astringent powder is used upon the patient's skin. Also, because it is unwise to neglect customary simple precautions, towels are employed as usual about the wound.

XLI. EXPLORATORY PRINCIPLE AND TECHNIQUE OF SECONDARY PLEUROTOMY.

By CARL BECK, M. D.
NEW YORK

As long as surgical diseases are still treated internally we will have to take "correcting operations" into account. Secondary rib resection illustrates one of the many attempts at correction, which could almost always be avoided by doing a timely primary resection. Closely related to the internal treatment of pyothorax is the therapy of aspiration, which is advised after the principle of "Wash my fur but don't make me wet," and which forms the main contingent for secondary rib resection. The postulate that each pleural empyema should be opened as soon and as widely as possible by resecting a rib without considering the nature of its precursor or its causation does scarcely meet with any objection at the hands of the surgical profession to-day. Among the reasons for these radical measures I would like to mention the presence, as I have also stated before, of fibrinous or cheesy clots (*N. Y. Med. Presse,* Dec. 1886), which can only be removed through a wide opening. And this is only made possible by the resection of a rib.

It would go beyond the scope of this work were I to discuss some of these questions, which do not meet with any more opposition on the part of competent surgeons, considering only conditions due to injudicious treatment, in rare cases due to disease of the lungs, which is caused by a failure of the lung to adhere to the costal pleura. The layers of the pleura becoming fibrous after a long continuance of the inflammatory irritation, the simple removal of several fragments of rib covering the empyema cavity is not sufficient. The costal pleura becomes so firm, that it represents an osseous coat-of-mail and therefore, as the genius of Schede has shown us, it must share the fate of the ribs, *i. e.,* it must be removed to the extent of its inelasticity. The technical appliances of this principle is concentrated in exposing the cavity through an incision reaching from the fourth rib, and, running in a curve, downwards to the posterior axillary line on a level with the tenth rib and then up again in a curved direction on the medial side of the shoulder blade. In this way access is gained to the largest cavities in empyema.

Although the procedure of Schede must be considered irreproachable and deserves the highest commendation, there are

some objections concerning his technique. First of all we must consider the fact that most of the cases of old pyothorax do not need so typical a procedure, in other words that Shede's method reaches beyond the mark. It is, in fact, one of the bloodiest and most exhaustive surgical proceedings. It offers indeed no little danger to the patient who is already weakened through a prolonged state of debility, and the operation of Schede should not only be performed by the agile hands of its inventor, but must also be carried out by the average surgeon. *Si duo faciunt idem, non est idem.* As a matter of fact modern surgery does not present a suitable field for the development of typical operations. Even the fundamental principles of incision for amputation, sacred for thousands of years, and formerly the *pièce de résistance* of the old masters, have become shaky, the surgeon generally adapting himself to the individuality of the case.

If we consider that old empyema-cavities show a many-sided picture, which even the all-penetrating Roentgen rays cannot faithfully portray, it becomes evident that a typical method of resection is only advisable in a minor number of cases. Of course we can measure the extent of the cavity by pouring in fluids, and the Roentgen rays give us a fair inlook after the infusion of iodoform glycerine which increases the shadows. It is far from being so distinct that we can found a detailed plan of resection upon it.

Thoughts of this character have induced me to follow a method of procedure which fits each individual case. It is found in the principle, to perfect the plan of operation while operating, that is to go ahead in an explorative measure.

The exploratory incision in cases of diseases of the pleura had already been taken up by me with good results in June, 1894, (see Exploratory Pleurotomy and Resection of Costal Pleura, *N. Y. Med. Journal*, June 15, 1895). It concerned a case in which osseous lumps had formed on both sides of the pleura after a long standing inflammatory process and had caused respiratory disturbances. It was reserved for the explorative resection of a rib in the region of dulness, not only to uncover this condition while proceeding slowly and gradually, but also to remedy it by the removal of the enormously thickened layers.

The strict attention to this principle has served me as a model for the secondary pleural incision, which I have described in an essay on Pyothorax, in the *International Medical Magazine*, January, 1897.

My method of proceeding is begun by resecting the rib which lies about in the middle of the roof of the cavity regardless of the pleural fistula. (Fig. 1 illustrates this.) The fistula is utilized for

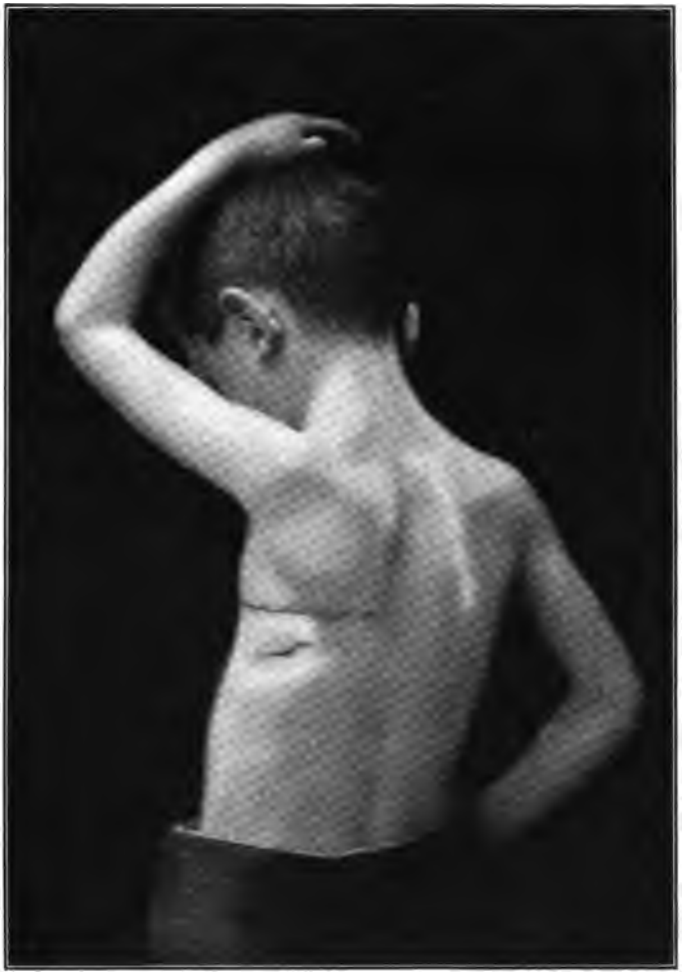

FIG. 1.
Resection of ribs and portion of scapula after the exploratory method.

the passage of a sound, but during the operation itself it is avoided, as in old cases osseous projections are formed which make the direct method harder and are more easily attacked from the side. The pleura underlying the resected rib is now incised. By means of a lateral incision enough room is gained to inspect a large part of the cavity and palpate the cavity walls. The use of the pleural speculum advised for primary resections (*Berliner Klin. Wochenschrift,* 1898, No. 15), is not necessary in such cases.

If the cavity is small and the patient is in a fairly good condition, which is the exception, then the next two or three ribs are resected from the vertical incision in proportion to the extent of the cavity beneath, while the soft parts are retracted with tenacula. The costal pleura is then excised by means of a blunt pointed knife. If the wheals are thick, then the intercostal arteries are so much diminished in lumen by compression that the hemorrhage can be regulated by temporary pressure. Then soft parts and ribs may be divided at the same time. Although this formation of wheals is to be most expected in very advanced cases, one should not rely too much upon such helps of nature, but make sure by means of a temporary prophylactic ligature *en masse* by the aid of a large aneurysm needle. In more extensive cases the ribs are divided successively in the same manner after having ascertained by palpation the presumptive length of each piece as it is incised. The palpation also tells whether the pleura beneath the ribs is elastic or must also be sacrificed. The incision of the soft part proceeds likewise, which results in a very irregular looking flap. But no particular attention needs to be paid to the shaping of the flap, as it must depend more or less upon that of the cavity. Accordingly I do not mind cross-incisions either. If the scapula is found to be in the way a portion of it is excised.

A mentionable point, which has to my knowledge not been presented in literature and is not of rare occurrence, is the concave arrangement of the lung surface, which overbridges a certain amount of the cavity. The lateral parts of the pulmonary pleura succeeded here to attach themselves to the costal pleura, fibrous adhesions holding them there. But the middle portion did not follow and now forms the floor of the cavity over which the approximated sides create the roof. We could compare the picture of this remarkable condition with that when one presses in the lung surface with the thumb so far that the sides of that segment of the lung collapse in a ring around it. This resembles the longitudinal fold formed of the gastric wall which is united in Witzel's gastrostomy over a tube so that a canal is made of it.

Great precaution is especially necessary at the beginning of the operation. If we were to cut in directly without first exploring the cavity thoroughly after the resection of the first rib then we surely would wound the lung. But if we have located the place of the thin portion where the pulmonary pleuræ join, then it is possible to finish the process by blunt dissection after having carefully divided the fibrous tissues. The lateral portions then gape apart and the circular cavity is transformed into a flat one. A partial decortication is then indicated. It appears that the right half of the lung is particularly prone to this sort of adherence, due perhaps to the presence of the middle segment, and which represents a desire to heal in the wrong direction.

Poor results are often traced to the fact that old empyemas show a predilection for the scapular region. And just here a thorough "decostation" is indispensable.

Similar views apply to the apex of the pleural cavity, which is not touched by Schede's procedure. Here it is also only the removal of the cavity-roof which makes a sore possible. The fear of wounding this region so plentiful in blood vessels seems to be responsible for the fact that one is loth to draw the upper ribs into the region of operative measures. The following procedure, which has yielded me much service in the resection of the point of the costal dome which covered the pleural space, I would like to recommend on account of its safety.

With the arm at right angles the incision is led close to the lower border of the pectoralis major in a horizontal direction till it ends at the lower part of the anterior margin of the deltoid muscle. The muscles are then dissected back superiorily, until the axillary region is free. The vessels and muscles are grasped by strong blunt hooks and pulled upward. At times separation is possible only by the aid of lateral incisions into both pectorales. Thus the vessels are temporarily set *hors de combat* and the ribs can be removed according to the indicated principles. If it is very difficult to reach the first rib by means of the pectoro-axillary incision, then the clavicle is resected temporarily.

The decorticated flap is then trimmed and placed on the pulmonary surface of the pleura. If pieces of the pulmonary pleura have been removed agglutination is much easier. In my afore mentioned monograph (Jan., 1897) I laid particular stress upon the fact that the callous areas of the pulmonary pleura should also be removed. This is not easy in debilitated patients, but whenever possible the operation should be finished at one sitting, for at a later supplementary operation we must partly destroy the fruits of the first. Of course in a case of doubt we must sacrifice them for the patient's life. This kind

of decortication we gain by flat, saw-like cutting procedures similar to the method of preparing microscopical sections. The principle is akin to that of Delorme and Fowler, but is not practised methodically as advised by these authors, and only as the necessity arises after the exploratory section and then as a supplement to the resection of the costal pleura.

As I see from a later publication Jorden (*Beiträge zur Klin. Chir.*, *Bd.* 34) and Krause (the same, Bd. 24, v. 1) likewise report good reports from this combination.

For dividing the rib I use an annular periostome and an elevatorium rib shears. The first is a sort of aneurysm needle, but flat, and formed to embrace the rib, as I might say. After

Fig. 8.

the dissection of the periosteum it serves as a retractor. It is combined with an elevator. The shears can be taken apart, so that both halves can be applied separately as in the obstetrical

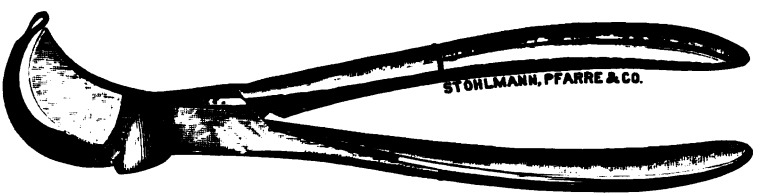

Fig. 9.

forceps. (The instruments are pictured in the *N. Y. Med. Record*, May 19, 1894, and the modern modifications in the *Journal of Surg. Technology*, Dec., 1900.)

It was obvious to apply the same principle to the treatment of lung abscess and therefore I recommended the exploratory pleuro- and pneumotomy in cases in which other methods, especially aspiration, gave no results. (See on the diagnosis and treatment of abscess of the lung, *N. Y. Med. Journal*, 28 Aug., 1897). Later Tuffier reached the same conclusion (Chirurgie des poumons, xii International Congress in Moscow, 1897). Of course the use of the Roentgen rays has greatly facilitated the process of locating the abscess. But the carrying out of the operative plan will have to shape its course in accordance with the principles pointed out.

In regard to anæsthesia I must remark that I never allow a full anæsthesia in a case of primary resection. Ether can not be considered on account of its congestive action on the lungs, and chloform endangers the heart displaced and compressed. Therefore I like to operate after the method of Schleich, after having anesthetized the point of puncture with ethyl chloride. But if I cannot do without chloroform I inject morphine beforehand and frequently interrupt the anæsthesia. In very low patients I use a prophylactic saline injection.

Fibrinous clots and necrotic fragments are removed at once, whether primary or secondary resection is performed. The edges of the cavity are packed with iodoform gauze. Immediately after the operation this is done tightly, later on loosely. Then drainage tubes, enveloped by iodoform gauze, may also be used. Thus irrigations are not only rendered unnecessary, but are in fact harmful.

During the after-treatment stress is to be laid upon early gymnastics, which favor expansion of the lungs. To this end I recommend dumb bells and practice on a bugle. The regeneration of the resected ribs is illustrated by the Roentgen rays (see skiagraphs in my previous publication on this subject).

IN MEMORIAM.

EDWIN MOTT MOORE, Permanent Member.
Died at Rochester, March 3, 1902, æt. 89.

MOSES T. BABCOCK, Permanent Member.
Died at Hammondsport, March, 1902, æt. 77.

THEODORE C. WALLACE, Permanent Member.
Died at Cambridge, March 12, 1902, æt. 72.

J. K. LEANING, Permanent Member.
Died at Cooperstown, April 4, 1902, æt. 79.

ABEL MIX PHELPS, Permanent Member.
Died at New York, October 6, 1902, æt. 51.

I. N. GOFF, Permanent Member.
Died at Cazenovia, October 13, 1902, æt. 69.

FRANK L. MARTINDALE, Permanent Member.
Died at Port Richmond, October 26, 1902, æt. 72.

THEODORE F. C. VAN ALLEN, Permanent Member.
Died at Albany, October 28, 1902, æt. 41.

DANIEL P. PEASE, Permanent Member.
Died at New York, December 7, 1902, æt. 45.

ADAM T. VAN VRANKEN, Permanent Member.
Died at Watervliet, January 19, 1903, æt. 52.

JOSEPH EASTMAN, Honorary Member.
Died at Indianapolis, Indiana, June 5, 1902, æt. 60.

RUDOLF VIRCHOW, Honorary Member.
Died at Berlin, Germany, September 5, 1902, æt. 81.

1. IN MEMORIAM

Edward Mott Moore, M. D., LL. D.

BORN IN RAHWAY, N. J., JULY 15TH, 1814; DIED IN ROCHESTER, N. Y., MARCH 3RD, 1902.

By WILLIAM S. ELY, M. D.
ROCHESTER

Dr. Moore was justly proud of his ancestry. His grandfather, Samuel Moore, was born in New Jersey, and during the Revolution moved to Nova Scotia where Lindley Murray Moore, the father of Dr. Moore, was born May 31st, 1778. Lindley Murray Moore is remembered as a distinguished teacher and preacher. He early moved to New Jersey and married Abigail Lydia Mott, Aug. 19th, 1813. Edward Mott Moore was their first child. In his father's school he was thoroughly drilled in grammar and logic, and laid the foundations of learning and morality upon which later he built his substantial character and broad culture.

Dr. Moore's father, mother and paternal grandfather belonged to the Society of Friends, and at the home of his aunt, Lucretia Mott, in Philadelphia, he was brought in relation with this noted woman who was of the same religious belief, and is remembered for her interest in the Anti-Slavery and Woman's Rights movements.

To the end of his life Dr. Moore showed the influence of the sterling intellectual and moral environment of his youth. After a thorough preliminary education he took a scientific course in the Rensselaer School at Troy, N. Y., preparatory to entering upon the study of medicine in the office of Dr. Anson Coleman of Rochester in 1835. The medical student of the modern medical school with a three or four years graded course knows little of the difficulties attending the study of medicine in the first half or three-fourths of the last century. Then the student was supposed to read medicine for one year in a doctor's office when he was not engaged in caring for the doctor's horse, or compounding medicines, or making the rounds with the doctor in his wagon or sleigh. Later he would attend two short winter courses of didactic lectures in a medical school in which the attempt was made to cover the whole field of medicine and surgery in five months. From Dr. Coleman's office Dr. Moore went to New York where he took one course at the College of Physicians and Surgeons. Afterwards he studied for two years at the Medical

Department of the University of Pennsylvania where he was graduated in 1838. At his death he was the oldest medical Alumnus of the university. For a year after graduating Dr. Moore was a resident physician at Blockley Hospital, Philadelphia, and afterwards resident physician at Frankfort Asylum for the Insane near Philadelphia. At Blockley in 1839 he studied by means of vivisections the action of the heart. In connection with Dr. Pennock he made valuable experiments confirming Harvey's original observation that at the time of contraction of the heart the ventricle elongates. In 1840 Dr. Moore came to Rochester to begin his life work, bringing with him the first Stethoscope ever used in Monroe county. His inherited talents, his thorough early training, his medical education and hospital experience—the best then obtainable—made him well fitted for entering upon the practice of medicine and surgery in the then prosperous and rapidly growing Flour City of the Genesee Valley. He early showed a predilection for surgery, and though he never relinquished medical practice, he preferred to be known as a surgeon. His accurate knowledge, his temper and temperament and his self-confidence combined to strengthen his surgical tendencies, and to the end of his career he was one of the most noted surgeons of the state of New York. Beginning practice before the introduction of anæsthsia he performed all of the capital operations without its aid.

Within four years after he was graduated Dr. Moore was chosen Professor of Surgery at Woodstock, Vt. Here and at Pittsfield, Mass., he lectured for eleven years. In 1854-5-6 he filled the Chair of Surgery at Starling Medical College, Columbus, Ohio, and in 1858 his increasing reputation as a teacher of surgery brought him the appointed of Professor of Surgery in the Buffalo Medical College, a chair which he continued to hold until 1883. He was a noted authority on the treatment of fractures and dislocations, and made valuable additions to these branches of surgery. His contributions to the pathology of Colles's fracture and fractures and dislocations of the clavicle will always have a great historic value, and in many cases the treatment for these injuries which Dr. Moore originated will be found perfect.

As a lecturer he spoke without notes, and was distinguished for the logical presentation of his subject with a style, clearness and force that have seldom been equalled by any public speaker. For over forty years he taught correct views of surgical procedures to thousands of students, many of whom acknowledged their indebtedness to him for that subtle inspiration in their work which only a living master of his art can impart.

When not engaged in teaching Dr. Moore was in active practice in Rochester and had a large consulting practice over a wide district of territory. He became a permanent member of the Medical Society of the State of New York in 1870, and its subsequent transactions bear witness to the activity of his mind. His original papers and his valuable discussions are noted in numerous volumes of the society's proceedings.

To preventive medicine Dr. Moore gave much time and thought. His reputation grew steadily until he was the recipient of a great number of medical honors. Apart from the chairs which he filled in medical colleges he was successively chosen president of the Monroe County Medical Society, of the Rochester Medical Society, of the Central New York Medical Society, of the Medical Society of the State of New York, of the Medical Association of the State of New York, of the American Surgical Association, of the American Medical Association, of the State Board of Health, and was a delegate to the International Medical Congress at Copenhagen in 1894. He was an honorary member of the Rochester Pathological Society, the Rochester Academy of Medicine, and the Hospital Medical Society of Rochester. In these numerous official position he became widely known by the medical profession of this country and of Europe, and the honors which came to him were the just recognition by his professional associates of his distinguished abilities.

But there is another side to the character of Dr. Moore which deserves consideration—we refer to his civic character. With him the humanities of life were not limited to the practice of medicine and surgery. His mind was too great not to see the broad field for philanthropic work which lay outside his immediate profession. So he early became identified with many altruistic movements, and at his death was by unanimous consent the leading citizen of Rochester. Early foreseeing the need of a system of public parks for Rochester, he outlined what such a system would mean for its growing population. Gradually the need grew upon his mind until he felt that the time had come for the project to take practical form. Then a strong and persistent agitation for public parks was begun and carried on against widespread opposition until the necessary legislation was secured. In 1888 he was chosen first president of the park commission, a position which he held until his death. Under his direction and with the aid of leading citizens, the present system of public parks in Rochester was elaborated. He lived to see the realization of his long cherished idea, and his greatest pleasure in his declining years was to note the appreciative recognition of his efforts by the thousands of young and old who found

their recreation and inspiration in visiting the parks. They are his living monument, and will be so considered by all historians of Rochester.

In all measures for the advancement of education Dr. Moore took an active interest. He was chosen a trustee of the University of Rochester in 1871. From 1886 to 1893 he was Vice-President of its Board of Trustees, and after 1893 and until his death President, giving for thirty years to the university much of his thought and time. In 1870 the university conferred on him the degree of Doctor of Laws. He was a charter member of the Board of Trustees of the Reynolds Library, and from 1890 until his death was President of the board. As the President of the Red Cross Society of Monroe County from 1881 to his death he manifested an interest in all efforts for the relief of the miseries of war, and for the mitigation of the suffering and distress by the calamities of nature.

Dr. Moore's life covered a long and eventful period. He saw wonderful changes wrought out in every department of human activity. To the last days of his life his mind was clear, and his interest in every scientific movement was unabated. To the city in which he had lived for nearly seventy years he was strongly attached. His professional eminence, benignant personality, grace of manner and devotion to beneficient objects were recognized by all who knew him, and he died full of years and honors.

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2. MEMORIAL ADDRESS

Abel Mix Phelps, M. D.
OF NEW YORK

By FRANK VAN FLEET, M. D.
NEW YORK

It is a melancholy task to write a memoir of a friend, and yet one not unmixed with pleasure. It is sad to think of death as an event which severs earthly ties and friendships; if one thinks of it only in this light there can be nothing but grief in connection with it. To one who believes in a future life death is not the end, but the beginning, and while we mourn in our selfishness that a friend has left us, yet we rejoice that the one who has gone has been freed from the cares and troubles which beset us here and has been allowed to enter upon the life eternal.

At our time of life the wild grief of youth at the loss of friends and dear ones gives way to feelings of sorrow and loneliness, for one by one our friends depart and our circle narrows until we

count those who are left, who become dearer to us than ever. As Young says:

> "Men drop so fast, ere life's mid-stage we tread,
> Few know so many friends alive, as dead."

Abel Mix Phelps was born at Alburg Springs, Vermont, January 27, 1851. He was descended from an English family which settled in Connecticut in 1630, a family which has given a number of eminent men to this country, among whom we may name the Hon. Edward J. Phelps, at one time Minister to England, and the Hon. William Walter Phelps, late Minister to Germany.

It is a coincidence that Dr. Phelps, had he lived, would have been fifty-two years of age to-day, and it is fitting that this Society he loved so well should devote a portion of its valuable time in paying a tribute to his memory, on this, the anniversary of his birth.

Dr. Phelps began his medical career at an early period of his life, having graduated in medicine from the University of Michigan in 1873. His position in the front ranks of medicine is too well known to require extended argument on my part. It is probable that many who listen to me to-day are more familiar with his work than the writer of this memoir.

He became an active, progressive surgeon from the beginning, and one whose opinions were eagerly sought by those who needed the advice he was competent to give. His position as surgeon to railroad and mining companies, while still a very young man, gave him an experience which, coupled with his intelligence and great good sense, made his opinion extremely valuable as an expert, and he was frequently called upon in this capacity.

It was as an Orthopedic Surgeon that he was especially known among his professional brethren, and it was said of him, by those most familiar with his work, that for originality of ideas, force of execution and intrepidity of action he was indeed a genius. As a medical teacher he had few equals, and it was gratifying to his intimate acquaintances to see the eagerness with which the large classes it was his pleasure to lecture to in the Post-Graduate College, listened to his clinical demonstrations. His was the force and power which made useful citizens out of many crippled children in the twenty-nine years he practiced his profession, and many are the medical men living to-day who have gone from the benches in the lecture room to carry out the instruction given by this man; in every state in the Union are those who have been benefited and cured as the result of the working of that fertile brain.

Not only was Dr. Phelps known in this country as a leader in his chosen specialty, but abroad had his fame been heralded. After his graduation he practiced in this country for seven years,

and then went to Germany and spent four years in the company of the leading surgeons of the old world, studying their methods and exchanging ideas with them. America was the home of orthopedic surgery, and Dr. Phelps was an American all through. So highly was he thought of abroad that at a later period, when he again visited Europe, he made quite a triumphant tour, being feted on all sides, delivering lectures and performing operations in many of the German universities. An indication of how he was looked upon abroad was given by the remarks of the Vienna surgeon, Dr. Adolph Lorenz, during his recent trip to this country. Dr. Lorenz said that one of the pleasures he had looked forward to in his visit to this country was that of meeting his friend, Dr. Phelps, and the news of his death had marred what would otherwise have been a trip of unbroken pleasure.

At home the profession recognized Dr. Phelps' worth by giving him positions of honor, as well as in more substantial ways. He held the positions of Professor of Surgery in the University of Vermont, and of Professor of Orthopedic Surgery in the Medical Department of the University of New York and in the New York Post-Graduate Medical School and Hospital.

Dr. Phelps had a wide circle of acquaintances among medical men, and laymen as well. It was the speaker's honor to be chairman of his business committee when he was President of this Society, and it is a matter of record the number of eminent men who journeyed from other states to this our capital city, to read papers and take part in discussions, at his request. The meeting over which Dr. Phelps presided was one of the most successful this Society ever had, and I am in the position to say that it was due entirely to his efforts.

Dr. Phelps had two loves in the profession, and to each he gave his best efforts. They were the Medical Society of the State of New York and the New York Post-Graduate Medical School and Hospital. For the latter he worked along the lines of developing the Orthopedic Department, of which he was the head. In the early days of that institution, when it had few students, little money and not an abundance of clinical material, Dr. Phelps sent around among the tenement houses, hunting up crippled and deformed children, giving them, of course, gratuitous treatment, and paying for their necessary appliances. He lived to see his department rank, with the rest of the institution, with the best of its kind in the land, happy in the knowledge that he was an important factor in its success. The good his work in this particular has done humanity is, in itself alone, evidence of the value of his life to the community.

To this State Society and to the Medical Society of the County of New York our friend was indeed a pillar of strength. He

was proud of this system of societies' long period of usefulness, and he looked upon its record of honorable service to the state of New York, of over ninety-six years when he died, as one of the grandest possessions of the medical profession in this country. He was anxious that the hundredth anniversary of its incorporation by the state legislature should be fittingly commemorated. His was the mind which played an important part in bringing about the appointment of the committee whose purpose it is to effect a union of the opposing elements in the medical profession in this state. He saw the possibility of this occurring in time for our centennial celebration, when a strong medical body of three thousand or more members would start anew the work of uplifting the profession and protecting the public health.

Dr. Phelps was not a man to sacrifice principle or honor. His idea was to make honest and straightforward overtures to the New York State Medical Association looking toward an amalgamation. He saw, also, the possibility of these overtures failing, and in that event he advocated going straight to the American Medical Association and asking just treatment for our State Society, that we be accorded the same recognition given to Massachusets, Mississippi, Illinois and Iowa, these four states refusing to adopt the Code of Ethics of the American Medical Association, and still being recognized by that body. Failing in this, Dr. Phelps and the speaker were agreed that another national body should be created, fully cognizant of the fact that this would entail the expenditure of both time and money. Dr. Phelps was a man of resources and nothing daunted him. He suffered defeat at times, but arose equal to the emergency and wrested victory where many men would have considered failure as inevitable and final.

It was the speaker's privilege at the annual meeting of this Society in 1902 to share a hotel room with Dr. Phelps, and in the dining room during the two days we spent in Albany we had as companions two of our state senators and an ex-governor of Vermont, who were boyhood companions of our deceased friend. It was interesting to hear them recount the stories of their boyhood days, and from them I gathered that the boy was father to the man. As a boy he could outdistance his companions in running, skating and jumping, and in all their sports he showed the same leadership which clung to him through life. They respected him, and with them, as with his associates of later life, he left his imprint, and all were better for having known him.

In the writings of Omar Khayyam we find these lines:

"The world will turn when we are earth
As though we had not come or gone;
There was no lack before our birth,
When we are gone there will be none."

One cannot have known noble men such as has fallen to my lot to know, and feel that this is true. The world will turn, undoubtedly, after we are gone, as it did before we came, but no person lives on this globe and leaves it exactly as it was before he came. His imprint is there, for good or for ill, and I am proud to stand here and say the influence of our friend was for good.

To me Dr. Phelps was the sincerest of friends. He had his many little peculiarities, some of them being very amusing, but they were the marks of the man. He interested and amused you while you admired his manly qualities. Generous to a fault, all that he posssesed was yours for the asking. He had a beautiful home, and his home life was of the loveliest character. He and his good wife were most hospitable, and many of us will recall the pleasant times we have spent as their guests. Together they almost worshipped their two infant daughters. To his family his loss is beyond estimation, and I am sure the sympathy of every member of this Society goes out to them in the fullest measure.

Dr. Phelps died Oct. 6th, 1902, in the prime of life and in the fullness of his manhood. To us it seems that if he had lived longer he would have continued to be of benefit to his race, but we do not know the wisdom of that power which is greater than ours. In the life Dr. Phelps lived he accomplished great things; many of us will live longer and achieve less. He lived a rapid life and reached an early end. When the record is finally written up, it will doubtless be determined that he did the work laid out for him to do, and did it well. Others will take up the work where he left it, and so complete the chain. It is a beautiful system, this progressive life of ours, and its intricacies and mysteries should make us charitable to each other, and not too eager to find and enlarge each other's faults. A man seldom lives to complete the work he starts out to do in youth, but if he erects one stone on which succeeding generations gradually build the perfected structure, he has not lived in vain. My final thought in what seems to us the untimely death of our friend can be summed up in these lines of an unknown author:

"Nor shall we die at all
Till all our work is done;
God surely will not call
From earth a single one
Whom He has sent to do
Some special work, the plan
Of which, with Him, it would not do
To make it known to man.
Yet, 'tis enough to know
That we our race must run;
Man is immortal here below
Till all his work is done."

MEDICAL SOCIETY OF THE STATE OF NEW YORK

OFFICERS OF THE SOCIETY FROM ITS ORGANIZATION TO THE PRESENT TIME.

Year.	*President.*	*Vice-President.*	*Secretary.*	*Treasurer.*
1807	Wm. McClelland,	Alexander Shelden,	John Stearns,	Moses Willard.
1808	Nicholas Romayne,	Alexander Shelden,	John Stearns,	James G. Graham.
1809	Nicholas Romayne,	Alexander Shelden,	John Stearns,	Andrew Proudfit.
1810	Nicholas Romayne,	Alexander Shelden,	John Stearns,	Andrew Proudfit.
1811	William Wilson,	Westel Willoughby,	Benj. R. Bevier,	Asa B. Sizer.
1812	John R. B. Rodgers,	Westel Willoughby,	John Stearns,	Asa B. Sizer.
1813	John R. B. Rodgers,	Joseph White,	John Stearns,	James L. Van Kleeck.
1814	John R. B. Rodgers,	Joseph White,	Ely Burritt,	James L. Van Kleeck
1815	Joseph White,	Ely Burritt,	James Low,	Westel Willoughby.
1816	Joseph White,	Ely Burritt,	James Low,	John Stearns.
1817	John Stearns,	Henry Mitchell,	James Low,	Chas. D. Townsend.
1818	John Stearns,	Henry Mitchell,	Peter Wendell,	Chas. D. Townsend.
1819	John Stearns,	Westel Willoughby,	Chas. D. Townsend,	John Downs.
1820	John Stearns,	Westel Willoughby,	Chas. D. Townsend,	John Downs.
1821	Samuel L. Mitchell,	Peter C. Adams,	Chas. D. Townsend,	John Downs.
1822	Samuel L. Mitchell,	Thomas Fuller,	Chas. D. Townsend,	T. Romeyn Beck.
1823	Alexander Coventry,	John H. Steele,	Chas. D. Townsend,	Jonathan Eights.
1824	Alexander Coventry,	John H. Steele,	Chas. D. Townsend,	Jonathan Eights.
1825	James R. Manley,	T. Romeyn Beck,	Platt Williams,	Jonathan Eights.
1826	James R. Manley,	T. Romeyn Beck,	Platt Williams,	Jonathan Eights.
1827	T. Romeyn Beck,	Laurens Hull,	Platt Williams,	Jonathan Eights.
1828	T. Romeyn Beck,	Laurens Hull,	Platt Williams,	Jonathan Eights.
1829	T. Romeyn Beck,	Henry Mitchell,	Joel A. Wing,	Jonathan Eights.
1830	Jonathan Eights,	Henry Mitchell,	Joel A. Wing,	Platt Williams.
1831	Jonathan Eights,	Thomas Spencer,	Joel A. Wing,	Platt Williams.
1832	Thomas Spencer,	James McNaughton,	Joel A. Wing,	Platt Williams.
1833	Thomas Spencer,	James McNaughton,	Joel A. Wing,	Platt Williams.
1834	John H. Steele,	James McNaughton,	Joel A. Wing,	Platt Williams.
1835	John H. Steele,	James McNaughton,	Joel A. Wing,	Platt Williams.
1836	James McNaughton,	Samuel White,	Joel A. Wing,	Platt Williams.
1837	James McNaughton,	Samuel White,	Peter Van O'Linda,	Platt Williams.
1838	Laurens Hull,	Sumner Ely,	Peter Van O'Linda,	Platt Williams.
1839	Laurens Hull,	Sumner Ely,	Peter Van O'Linda,	Platt Williams.
1840	Sumner Ely,	John B. Beck,	Peter Van O'Linda,	Platt Williams.
1841	John B. Beck,	William Taylor,	Peter Van O'Linda,	Platt Williams.
1842	William Taylor,	Joel A. Wing,	Peter Van O'Linda,	Platt Williams.
1843	Samuel White,	Joel A. Wing,	Peter Van O'Linda,	Platt Williams.
1844	Joel A. Wing,	Harman Van Dusen,	Peter Van O'Linda,	Platt Williams.
1845	James Webster	Robert G. Frary,	Peter Van Buren,	Peter Van O'Linda.
1846	John McCall,	Stephen Hasbrouck,	Peter Van Buren,	Peter Van O'Linda.
1847	Thos. W. Blatchford,	Alex. Thompson,	Peter Van Buren,	Peter Van O'Linda.
1848	Alex. H. Stephens,	Alex. Thompson,	Peter Van Buren,	Peter Van O'Linda.
1849	Alex. H. Stephens,	Alex. Thompson,	Peter Van Buren,	Peter Van O'Linda.
1850	Alex. Thompson,	Jenks S. Sprague,	Thomas Hun,	Peter Van O'Linda.
1851	Robert G. Frary,	Jenks S. Sprague,	Thomas Hun,	Peter Van O'Linda.

Year.	*President.*	*Vice-President.*	*Secretary.*	*Treasurer.*
1852	Alonzo Clark,	Jenks S. Sprague,	Thomas Hun,	Peter Van O'Linda.
1853	Jenks S. Sprague,	Peter Van Buren,	James H. Armsby,	Peter Van O'Linda.
1854	Charles R. Coventry,	Peter Van Buren,	Howard Townsend,	Peter Van O'Linda.
1855	Frank H. Hamilton,	Thomas Hun,	Howard Townsend,	Peter Van O'Linda
1856	Alden March,	Chas. S. Goodrich,	Howard Townsend,	J. V. P. Quackenbush.
1857	Augustus Willard,	Thos. C. Brinsmade,	Sylves. D. Willard,	J. V. P. Quackenbush.
1858	T. C. Brinsmade,	George W. Bradford,	Sylves. D. Willard,	J. V. P. Quackenbush.
1859	B. Fordyce Barker,	Daniel T. Jones,	Sylves. D. Willard,	J. V. P. Quackenbush.
1860	Daniel T. Jones,	Edward H. Parker,	Sylves. D. Willard,	J. V. P. Quackenbush.
1861	Edward H. Parker,	Andrew Van Dyck,	Sylves. D. Willard,	J. V. P. Quackenbush.
1862	Thomas Hun,	Daniel P. Bissell,	Sylves. D. Willard,	J. V. P. Quackenbush.
1863	Daniel P. Bissell,	Joel Foster,	Sylves. D. Willard,	J. V. P. Quackenbush.
1864	Frederick Hyde,	George J. Fisher,	Sylves. D. Willard,	J. V. P. Quackenbush.
1865	Henry W. Dean,	Jos. C. Hutchison,	William H. Bailey,	J. V. P. Quackenbush.
1866	Joseph C. Hutchison,	Julian T. Williams,	William H. Bailey,	J. V. P. Quackenbush.
1867	John P. Gray,	Lake I. Teft,	William H. Bailey,	J. V. P. Quackenbush.
1868	J. V. P. Quackenbush,	James P. White,	William H. Bailey,	John V. Lansing.
1869	James P. White,	George Burr,	William H. Bailey,	John V. Lansing
1870	S. O. Vander Poel,	Gilson A. Dayton,	William H. Bailey,	John V. Lansing.
1871	William C. Wey,	Andrew F. Doolittle,	William H. Bailey,	Charles H. Porter.
1872	Cornelius R. Agnew,	B. F. Sherman,	William H. Bailey,	Charles H. Porter.
1873	Edward M. Moore,	Francis Burdick,	William H. Bailey,	Charles H. Porter.
1874	George J. Fisher,	Harvey Jewett,	William H. Bailey,	Charles H. Porter.
1875	Thos. F. Rochester,	Ellsworth Eliot,	Edward R. Hun,	Charles H. Porter.
1876	E. R. Squibb,	J. V. Kendall,	Edward R. Hun,	Charles H. Porter.
1877	J. Foster Jenkins,	A. L. Saunders,	W. Manlius Smith,	Charles H. Porter.
1878	D. B. St. John Roosa,	Judson C. Nelson,	W. Manlius Smith,	Charles H. Porter.
1879	Henry D. Didama,	Nath. C. Husted,	W. Manlius Smith,	Charles H. Porter.
1880	William H. Bailey,	Abraham Jacobi,	W. Manlius Smith,	Charles H. Porter.
1881	Abraham Jacobi,	William Govan,	W. Manlius Smith,	Charles H. Porter.
1882	Harvey Jewett,	E. D. Ferguson,	W. Manlius Smith,	Charles H. Porter.
1883	Alexander Hutchins,	H. G. P. Spencer,	W. Manlius Smith,	Charles H. Porter.
1884	B. F. Sherman,	P. R. H. Sawyer,	W. Manlius Smith,	Charles H. Porter.
1885	A. Vander Veer,	Alfred C. Post,	W. Manlius Smith,	Charles H. Porter.
1886	William S. Ely,	Sol. Van Etten,	W. Manlius Smith,	Charles H. Porter.
1887	Alfred L. Loomis,	A. M. Phelps,	W. Manlius Smith,	Charles H. Porter.
1888	Samuel B. Ward,	A. Walter Suiter,	W. Manlius Smith,	Charles H. Porter.
1889	Daniel Lewis,	Alfred Mercer,	Frederic C. Curtis,	Charles H. Porter.
1890	W. W. Potter,	L. S. Pilcher,	Frederic C. Curtis,	Charles H. Porter.
1891	A. Walter Suiter,	W. W. Crandall,	Frederic C. Curtis,	Charles H. Porter.
1892	Lewis S. Pilcher,	Henry L. Elsner,	Frederic C. Curtis,	Charles H. Porter.
1893	Herman Bendell,	C. L. Stiles,	Frederic C. Curtis,	Charles H. Porter.
1894	George Henry Fox,	Frank S. Low,	Frederic C. Curtis,	Charles H. Porter.
1895	Roswell Park,	William Maddren,	Frederic C. Curtis,	Charles H. Porter.
1896	James D. Spencer,	L. Duncan Bulkley,	Frederic C. Curtis,	Charles H. Porter.
1897	Seneca D. Powell,	Lucien Howe,	Frederic C. Curtis,	Charles H. Porter.
1898	John O. Roe,	E. F. Brush,	Frederic C. Curtis,	Charles H. Porter.
1899	Willis G. Macdonald,	John Gerin,	Frederic C. Curtis,	Charles H. Porter.
1900	A. M. Phelps,	George Seymour,	Frederic C. Curtis,	O. D. Ball.
1901	Henry L. Elsner,	Louis N. Lanehart,	Frederic C. Curtis,	O. D. Ball.
1902	Henry R. Hopkins,	William A. Moore,	Frederic C. Curtis,	O. D. Ball.
1903	A. T. Bristow,	Edward B. Angell,	Frederic C. Curtis,	O. D. Ball.

OFFICERS, 1902

PRESIDENT,

ALGERNON T. BRISTOW, Brooklyn.

VICE-PRESIDENT,

EDWARD B. ANGELL, Rochester.

SECRETARY,

FREDERIC C. CURTIS, Albany.

TREASURER,

O. D. BALL, Albany.

MEMBERS.

I. DELEGATES FROM COUNTY MEDICAL SOCIETIES, ETC.

(By-laws, Chapter I.)

Albany County:
Robert Babcock, Albany.
E. A. Bartlett, "
George Blumer, "
T. L. Carroll, "
A. W. Elting, "
C. H. Moore, "
W. H. Murray, "
Thomas A. Ryan, "
G. E. Lochner, "
M. MacHarg, "
C. H. Richardson, "
A. H. Traver, "
J. W. Wiltsie, "
Arthur Sautter, "
H. L. K. Shaw, "
Clement F. Theisen, "
E. A. Vander Veer, "
W. B. Sabin, Watervliet.
J. L. Archambault, Cohoes.
Daniel C. Case, Slingerlands.

Allegany County:
O. E. Burdick, Little Genesee.
Frank E. Howard, Friendship.
H. L. Hulett, Allentown.
W. H. Loughead, Nile.
George W. Roos, Wellsville.

Broome County:
William E. Ard, Binghamton.
Richard R. Daly, "
Edward F. Day, "
J. Killen, "
Frederick W. Miller, "
W. S. Overton, "
Frank W. Sears, "
Edward L. Smith, "
Charles R. Seymour, "

Cattaraugus County:
F. C. Beals, Salamanca.
M. C. Hawley, East Randolph.
W. B. Johnston, Ellicottville.
A. D. Lake, Gowanda.
J. E. K. Morris, Olean.
Cassar Smith, "
James A. Taggert, Salamanca.
Edward Torrey, Alleghany.
W. H. Vincent, Hinsdale.

Cayuga County:
A. L. Hall, Fairhaven.
Sheldon Voorhees, Auburn.
M. B. Van Buskirk, "
Ledra Heazlit, "
D. A. White, Montezuma.

Chautauqua County:
C. E. Ellis, Sherman.
E. S. Rich, Kennedy.

Chemung County:
A. H. Baker, Elmira.
Arthur W. Booth, "
La Rue Colegrove, Wellsburg.
Frank W. Ross, Elmira.
Anna M. Stuart, Elmira.

Chenango County:
Robert H. Phelps, Norwich.
E. L. Gaus, Oxford.
Homer E. Smith, Norwich.
B. A. Harris, "
Thurston G. Packer, Smyrna.

Clinton County:
M. D. Briggs, Champlain.
Robert B. Lamb, Dannemora.
Cassius D. Silver, Plattsburgh.
T. A. Rodgers "
J. W. Hackett, Champlain.

Columbia County:
N. D. Garnsey, Kinderhook.
Henry W. Johnson, Hudson.
Martin M. Kittell, Kinderhook.
Emmett Niver, Hillsdale.
J. E. Vigeant, Elizaville

Cortland County:
H. S. Braman, Homer.
H. C. Hendrick, McGraw.
Benjamin Kinyon, Cincinnatus.
Frank D. Reese, Cortland.
M. R. Smith, McGraw.

Delaware County:
S. E. Churchill, Stamford.
Gilbert T. Scott, Davenport.
Stanton Hendrick, Treadwell.
George C. Smith, Delhi
Lloyd Palmer, East Meredith.

Dutchess County:
T. E. Bamford, Poughkeepsie.
Nelson Borst, "
E. M. Burns, "
J. H. Cotter, "
J. H. Doughty, Matteawan.
Isham G. Harris, Poughkeepsie.
C. W. Pilgrim, "
L. C. Wood, "
I. M. Cornell, Wappinger Falls.
G. H. Van Wagner, "

*Erie County:**
Justin G. Thompson, Angola.
John D. McPherson, Akron.
Walter D. Greene, 385 Jersey st
Henry D. Ingraham, 405 Franklin st
I. M. Snow, 476 Franklin st
Wm. G. Bissell, 143 Elmwood av
A. L. Benedict, 405 Franklin st.
N. W. Wilson, 153 Seventh st
Wm. C. Phelps, 146 Allan st
Edward H. Ballou, Gardenville
R. S. Meyers, Clarence Centre
F. E. Fronczak, 508 Fillmore av
Julius Ullman, 400 Franklin st
John H. Grant, 891 Ellicott sq
Wm. F. May, 658 Clinton st
C. E. Congdon, 1034 Jefferson st
Dewitt H. Sherman, 666 Main st
C. P. Smith, 90 N. Pearl st
G. R. Trowbridge, 1331 Main st
Geo. F. Cott, 16 Woodlawn av
F. E. L. Brecht, 361 William st
Jas. J. Mooney, 393 Seventh st
Joseph Fowler, 141 Delaware av
Chas. A. Wall, 306 Hudson av
J. Pohlman, 404 Franklin st
Benjamin G. Long, 520 Elmwood
Walden M. Ward, North Collins
J. H. Dowd, 378 Franklin st
J. B. Coakley, 339 Delaware av
Henry R. Lapp, Clarence
A. E. Woehnert, 155 Allen st
W. D. Bidaman, 330 Connecticut av
John J. Twohey, 301 Masten av
C. A. Clements, 916 Niagara av
E. M. Dooley, 406 Louisiana av
B. S. Bourne, Hamburg
W. G. Taylor, 822 Elmwood av
Harvey R. Gaylord, 568 Delaware av
Charles G. Stockton, 436 Franklin st
DeLancy Rochester, 409 Franklin st

Essex County:
——— ———

Franklin County:
C. A. Hastings, Constable

Fulton County:
William C. Wood, Gloversville
Charles B. Mosher, Johnstown
James K. Young, "
Nelson Everest, Gloversville
Frederick I. Jansen, Fonda

Genesee County:
——— ———

Greene County:
Ambrose Beach, Coxsackie
A. W. Van Slyke, "
L. B. Honeyford, Cairo
George Conkling, Durham
W. A. Wasson, Greenville

Herkimer County:
D. M. Devendorf, Herkimer
George S. Eveleth, Little Falls
Seymour S. Richards, Frankfort
U. G. Williams, Newport
Charles G. Strobel, Dolgeville

Jefferson County:
C. B. Forsythe, Alexandria Bay
James F. McCaw, Watertown
Chas. N. Bibbins, "
Gilbert D. Gregor, "
Fred'k B. Smith, "
Henry A. Hoyt, "
C. A. Drury, "
Elmer E. Eddy, Redwood
Geo. E. Sylvester, Black River
H. C. Potter, Morrisville

Kings County:†
R. B. Anderson, 765 Nostrand av
Henry F. Adams, 32 Palmetto st
A. S. Ambler, New Jersey
W. S. Applegate, 931 Flatbush av
Benjamin Ayers, 213 Jefferson st
F. R. Baker, 540 Bedford av

*Where no town is mentioned Buffalo is to be understood.
†All Brooklyn

L. G. Baldwin, 28 Schermerhorn st
Homer L. Bartlett, 88 Fennimore st
H. B. Bayles, 125 Seventh av
C. D. Beasley, 702 Greene av
Charles B. Bacon, Cumberland St. Hospital
Francis P. Beebe, 145 Clinton st
Nathan T. Beers, Jr., 1265 Bedford av
Wm. N. Belcher, 25 S. Portland av
Herman P. Bender, 683 Bushwick av
W. H. Biggam, 1197 Dean st
B. G. Blackmar, Ovington av
A. H. Bogart, 139 Seventh av
J. B. Bogart, 423 Washington av
R. C. Brewster, 126 Leffert's Pl
E. M. Bullwinkle, 80 E. Ninth st
W. L. Chapman, 19 LaFayette av
Carroll Chase, 191 Kingston av
G. W. Colby, 142 Putnam av
T. C. Craig, 187 Washington Pk
W. J. Cruikshank, 102 Ft. Greene Pl
Edward E. Cornwall, 146 Herkimer st
Henry P. DeForest, 369 Hancock st
George Drury, 235 Washington av
Wm. F. Dudley, 32 Livingston st
Benjamin Edson, 83 St. Mark's av
R. M. Elliott, L. I. State Hosp.
George Essig, 488 Bedford av
George Everson, 364 Greene av
H. A. Fairbairn, 249 McDonough st
G. N. Ferris, 910 Flatbush av
J. W. Fleming, 471 Bedford av
Louis N. Foote, 627 Marcy av
R. S. Fowler, 301 De Kalb av
Homer E. Fraser, 18 S. Portland av
W. M. Friend, 404 Clinton st
J. F. Golding, 363 Franklin av
Charles H. Goodrich, 280 Park Pl
W. H. Haynes, 401 Bainbridge st
J. C. Hancock, 43 Cambridge pl
Charles C. Henry, 56 Clark st
Burt D. Harrington, 34 Lenox rd
H. N. Hoople, 253 Gates av
Alexander C. Howe, 311 Cumberland st
John Horni, 179 Penn st
William S. Hubbard, 97 Halsey st
Clarence R. Hyde, 126 Joralemon st
J. W. Ingalls, 874 LaFayette av
A. M. Judd, 188 Sixth av
P. C. Jamison, 139 Montague st
William A. Jewett, 382 Adelphi st
Oswald Joerg, 12 Schermerhorn st
Florence Leigh-Jones, 508 3d st
Charles R. Jenkins, 31 Conselyea st
J. A. Kene, 64 Greene av
C. Le G. Kerr, 97 Cumberland st
C. D. Kevin, 719 Halsey st
James J. Keyes, 226 17th st
Francis S. Kennedy, 462 Greene av.
George F. Little, 469 Clinton av
Stephen H. Lutz, 551 Madison st
Stewart Lewis, 13 Cambridge Pl
George F. Little, 469 Clinton av
Lefferts A. McClelland, 78 McDonough
L. C. McPhail, 161 Hicks st
E. H. Mayne, 8744 18th st
W. H. Maddren, 131 S. Oxford st
Charles G. Molin, 184 State st
Archibald Murray, 69 Remsen st
Charles D. Napier, 1273 Bedford av
Wm. A. Northridge, 21 Hanson Pl
E. L. Oatman, 82 Remsen st
B. Onuf, 99 Berkley pl
Frederick C. Paffard, 238 Clinton st
Earnest Palmer, 155 Clinton st
Ralph H. Pomeroy, 511 Nostrand av
William P. Poole, 147 Clinton st
Malcolm E. Parrott, 379 Jefferson av
J. P. Pendelton, 42 Seventh av
Alexander Rae, 117 Henry st
William G. Reynolds, 162 Halsey st
Wilbur L. Rickard, 262 Stuyvesant av
Rubert S. Royce, 211 Greene av
C. T. Sauer, 284 6th av
F. W. Shaw, 327 Greene av
J. D. Shea, 427 8th st
Warren S. Shattuck, Jr., 147 Clinton st
W. S. Simmons, 338 Lafayette av
C. E. Schofield, 152 Taylor st
Wm. H. Sheppard, 202 52d st
Adam Schauf, 198 Vernon av
James A. Somers, 32 Lafayette av
John R. Stivers, 180 Lefferts pl
Charles H. Tag, 168 Keap st
D. E. Warren, State Hospital
R. W. Westbrook, 1145 Dean st
C. F. Yerdon, 1276 Herkimer st
Victor L. Zimmerman, 557 Halsey st

Lewis County:
A. C. Miller, Turin
P. H. von Zierolshofen, Croghan
F. E. Jones, Beaver Falls
F. A. Crane, Lowville
W. O. Hubbard, Lowville

Livingston County:
Wm. T. Shanahan, Sonyea
Annie M. Tremaine, "
F. J. Bowen, Mt. Morris
F. B. Dodge, "
John P. Brown, Nunda

Madison County:
E. H. Carpenter, Oneida
Charles H. Perry, "
C. M. Colegrove, Canastota
O. S. Langworthy, Hamilton
A. P. Dodge, Oneida

Monroe County:
Charles R. Barber, Rochester,
W. D. Becker, "
W. M. Brown, "
R. L. Carson, "
Robert G. Cook, "
C. E. Darrow, "
Simon L. Elsner, "
Wm. V. Ewers, "
C. A. Greenleaf, "
W. S. Hall, "
Loren W. Howk, "
C. A. Huber, "
Irving E. Harris, "
D. G. Hastings, "
J. M. Ingersol, "
Owen E. Jones, "
Frank A. Jones, "
C. T. La Moure, "
J. L. Roesboom, "
Nathan W. Soble, "
Benjamin Wilson, "
John Zimmer, "
W. J. Howe, Scottsville

Montgomery County:
W. J. Peddie, Fultonville
Otis Z. Bouton, "
George L. Meyer, Stone Arabia
James B. Conant, Amsterdam
Frederick I. Jansen, Fonda

New York County:
I. N. Love, 537 Fifth av
S. Alexander, 5 West 58th st
John Aspell, 105 Madison av
R. Bellamy, 210 West 57th st
W. Coleman, 5 West 30th st
R. S. Adams, 6 East 34th st
C. C. Barrows, 8 West 36th st
L. A. Conner, 53 East 49th st
C. W. Cutler, 36 East 33rd st
Geo. S. Dixon, 124 Lexington av
M. J. Echeverria, 141 W. 73rd st
F. S. Fielder, 33 West 93rd st
F. W. Gwyer, 130 East 38th st
Wm. T. Gibb, 55 West 38th st
Geo. D. Hamlen, 8 West 36th st
J. A. Hartwell, 50 West 53rd st
J. C. Johnston, 64 East 56th st
A. B. Johnson, 12 East 58th st
W. M. Polk, 7 East 36th st
O. H. Schultze, 1109 Madison av
G. Seeligman, 53 East 72nd st
Wm. Shannon, 117 West 81st st
Ed. P. Shelby, 30 West 32d st
Wm. F. Stone, 129 West 43rd st
Ben. T. Tilton, 59 West 36th st
Geo. Woolsey, 117 East 36th st
Wm. S. Stone, 1730 Broadway
R. T. Bang, 139 West 11th st
Reuben F. Monette, Manhattan State Hospital
Frank H. Magness, Manhattan State Hospital
Herman C. Evarts, Manhattan State Hospital
William K. Otis, 5 West 50th st
E. K. Dunham, 338 East 26th st
E. Le Fevre, 52 West 56th st
John H. Huddleston, 126 West 85th st
S. A. Brown, 11 West 45th st
Francis A. Scratchley, 154 East 72nd st
J. F. Erdman, 60 West 52nd st
C. G. Coakley, 11 West 45th st
H. M. Silver, 5 East 43rd st
J. E. Weeks, 136 West 16th st
C. H. Lewis, 47 West 58th st
E. H. Griffen, 112 West 45th st
B. Symonds, 410 West 20th st
R. C. James, 5 West 30th st
W. J. Pulley, 945 Madison av
S. Oppenheimer, 706 Madison av
Perry S. Boynton, 103 West 84th st
Henry F. Quackenboss, 159 West 88th st
Leon LeWald, 436 Lexington av
M. Nicoll, Jr., 168 West 48th st
G. R. Satterlee, 252 Madison av
John B. Huber, 41 West 45th st
A. W. Ferris, 343 Madison av
H. L. Reis, 219 East 12th st
J. K. Alvarez, 1159 Park av
Edward W. Peet, 348 Central Park West
Wm. B. Noyes, 28 West 61st st
Ludwig Weiss, 77 East 94th st
C. E. Lambert, 135 West 104th st
Henry Roth, 66 East 135th st
H. Koplik, 66 East 58th st
James H. Voorhees, 126 West 58th st
A. S. Vosburgh, 40 West 88th st
H. E. Hale, 77 West 50th st

L. W. Hotchkiss, 24 East 54th st
F. C. Wood, 205 West 57th st
H. A. Griffen, 346 Broadway
W. S. Bickham, 922 Madison av
David Bovaird, Jr., 126 West 58th st
E. L. Dow, 49 West 57th st
V. H. Norrie, 21 West 37th st
C. Wallace, 165 West 80th st
W. H. Caswell, 201 West 55th st
S. E. Jelliffe, 231 West 71st st
F. R. Oastler, 115 East 56th st
Charles H. May, 692 Madison av
W. K. Simpson, 952 Lexington av
Francis Huber, 209 East 17th st
Pearce Bailey, 4 West 50th st
Fred'k Peterson, 4 West 50th st
C. A. Whiting, 70 West 47th st
C. H. Peck, 37 West 48th st
T. H. Manley, 115 West 49th st
S. Tousey, 103 West 76th st
H. Cheesman, 171 West 91st st
F. J. C. Fitzgerald, 624 Lexington av
J. Milton Mabbott, 19 Fifth av
R. W. Eastman, 140 W. 76th st
Wm. O. Moore, 42 East 29th st
W. H. Porter, 210 West 57th st
J. R. Nilson, 69 West 50th st
G. M. Hammond, 58 W. 45th st
H. D. Chapin, 51 West 51st st
Leonard Weber, 25 West 46th st
Max Einhorn, 20 East 63rd st
H. B. Douglas, 121 East 36th st
S. G. Gant, 58 West 56th st
C. W. Allen, 30 East 33rd st
Franz Torek, 59 East 60th st
W. S. Bainbridge, 34 Gramercy Park
T. G. Lusk, 121 East 40th st
J. N. West, 71 West 49th st
S. S. Graber, 185 East 71st st
F. C. Combes, 165 East 116th st
J. S. Emans, 221 East 19th st
A. Rose, 126 East 29th st
J. B. Ferguson, 123 East 19th st
C. A. McWilliams, 50 E. 53rd st
C. R. L. Putman, 114 E. 38th st
F. N. Irwin, 10 West 39th st
J. J. McGrath, 105 East 73rd st
W. B. Brown, 214 West 44th st
F. N. Wilson, 106 West 47th st
D. H. Wiesner, 162 East 46th st
E. F. Smith, 324 West 46th st
Wm. H. Bates, 50 East 64th st
A. W. Hollis, 111 West 47th st
Wm. L. Culbert, 118 Madison av
G. H. Cobb, South Orange, N. J.
E. C. Dent, Manhattan State Hospital
L. A. Rodenstein, 561 West 162nd st
T. J. Harris, 117 East 40th st
C. A. Manson, 123 East 36th st
E. S. Thomson, 16 East 43rd st
John Van Doren Young, 60 West 76th st
W. W. Gilfillan, 24 West 59th st
H. Illoway, 1138 Madison av
C. N. Dowd, 135 West 73rd st
N. H. Henry, 48 West 11th st
E. P. Fowler, 18 East 58th st
A. B. Duel, 254 Madison av
Justin Wohlfarth, 119 West 129th st
Carl Koller, 715 Madison av
J. B. White, 1013 Madison av
R. A. Murray, 112 West 80th st
Alfred Meyer, 801 Madison av
J. B. Squier, 29 East 44th st
Morris Manges, 941 Madison av
F. J. Leviseur, 741 Madison av
M. McLean, 29 East 126th st
W. L. Stowell, 28 West 36th st
J. A. Kenefick, 29 West 36th st
Heinrich Stern, 56 East 76th st
W. P. Brandegee, 17 W. 46th st
J. B. Clemens, 19 East 38th st
W. B. Pritchard, 105 W. 73rd st
J. B. Rae, 668 East 157th st
S. M. Payne, 542 Fifth av
Wm. A. Macy, Willard, N. Y.
George Meyers, 47 East 61st st
S. A. Knopf, 16 West 95th st
L. Fischer, 65 East 90th st
J. B. Walker, 33 East 33rd st
P. J. McCourt, 233 West 23rd st
C. S. May, 205 West 57th st
J. L. Barnes, 16 East 43rd st
J. Brettauer, 45 East 64th st
W. A. Ewing, 134 West 58th st
J. H. Branth, 183 West 87th st
F. J. Parker, 49 West 50th st
Z. L. Leonard, 19 West 120th st
W. C. McFarland, 360 West 51st st
Frank H. McLaury, 244 West 42nd st
Franklin G. Ketcham, 318 West 84th st.
T. H. Willard, 1 Madison av
S. M. Brickner, 136 W. 85th st
S. F. Hallock, 36 East 65th st
Antonie P. Voilawsky, 72 St. Mark's Place
P. F. Sondern, 19 East 38th st
Frank N. Emmert, 110 West 132nd st
W. G. Le Boutillier, 45 West 50th st
L. W. Zwisohn, 1085 Lexington av

Homer Wakefield, 1048 Fifth av
John T. W. Rowe, Manhattan State Hospital

Niagara County:
F. J. Baker, Lockport
C. N. Palmer, "
Walter A. Scott, Niagara Falls

Oneida County:
B. P. Allen, Oriskany
Smith Baker, Utica
Thomas J. Bergen, Utica
Charles Bernstein, Rome
Frank D. Crim, Utica
Conway A. Frost, Rome
V. B. Hamlin, Clinton
C. R. Hart, New Hartford
LeRoy H. Jones, Utica
Wm. B. Roemer, "
L. F. Pattengill, "
Chas. P. Russell, "
D. H. Roberts, "
W. J. Schuyler, "
J. Nelson Teeter, Utica

Onondaga County:
H. C. Baum, Syracuse
J. H. Burch, Baldwinsville
B. F. Chase, East Syracuse
M. Helen Cullings, Syracuse
William A. Curtin, "
F. E. Easton, "
Hiram B. Hawley, "
A. W. Hedden, "
A. S. Hotaling, "
W. C. Kellogg, "
I. H. Levy, "
B. W. Loomis, "
C. E. McCrary, "
G. M. Price, "
A. B. Randall, "
Charles S. Roberts, "
F. W. Smith, "
W. J. Werfleman, "
I. M. Slingerland, Fayetteville
R. A. Whitney, Liverpool

Ontario County:
Walter B. Clapper, Victor

Orange County:
D. B. Hardenbergh, Middletown
W. J. Purdy, "
H. B. Swartwout, Port Jervis
H. Hardenbergh, "
C. E. Townsend, Newburgh
Wm. Hollinger, "
G. H. Brown, Highland Falls
Theo. Writer, Otisville
Jacob B. Peters, Walden
A. P. Farries, Florida

Orleans County:
R. W. Bamber, Kendall

Oswego County:
W. G. Babcock, Cleveland
H. G. Bartlett, Williamstown
Pascal M. Dowd, Oswego
Emory J. Drury, Phœnix
Leroy F. Hollis, Sandy Creek
Charles E. Low, Pulaski
H. P. Marsh, Fulton
George W. Nelson, Orwell
S. A. Russell, Fulton
C. A. Sheridan, Oswego

Otsego County:
A. W. Cutler, Oneonta
W. R. Lough, Edmeston
Charles E. Parish, Maryland
Henry D. Sill, Cooperstown
J. W. Swanson, Springfield

Putnam County:

Queens-Nassau Society:
J. H. Bogart, Roslyn
George H. Donahue, Northport
George W. Faller, Oyster Bay
Samuel Hendrickson, Jamaica
J. R. Hinkson, Long Island City
J. E. Hutcheson, Rockville Centre
Walter Lindsay, Huntington
R. F. Macfarlane, Long Island City
C. M. Niesley, Manhasset
E. H. Pershling, Lawrence
James D. Trask, Astoria
Wm. J. Burnett, Long Island City
P. H. Bumster, "
G. Herbert Carter, Huntington
F. T. de Lano, Rockville Centre

Rensselaer County:
C. H. Travell, Troy
M. A. Wheeler, "
Hiram Elliott "
Wm. Finder, "
William Kirk, Jr., Troy
Thomas G. Dickson, "
John B. Harvie, "
Thomas G. Wright, "
H. E. DeFreest, "
B. S. Booth, "
R. A. Kirkpatrick, "
A. D. Davidow, "
James Love, "

Richmond County:
Alfred H. Thomas, Clifton
William T. Van Vredenburgh, New Brighton
Jefferson Scales, New Brighton
John T. Sprague, Clifton
Wm. Bryan, W. New Brighton

Rockland County:
Frank E. Pagett, Spring Valley
S. Demarest, Suffern
E. B. Laird, Haverstraw
J. Senstacken, Stony Point
J. M. Hasbrouck, Garnerville

St. Lawrence County:
W. H. Hanbidge, Ogdensburg
H. A. McIlmoyl, "
James Wiltsie, Benson's Mines
S. E. Brown, Ogdensburg
Grant C. Madill, "
F. F. Drury, "
Daniel M. Taylor, Edwards

Saratoga County:

Schenectady County:
Alexander Ennis, Pattersonville
Charles F. Clowe, Schenectady
Charles G. Briggs, "
Joseph Raymond, "
Dayton L. Kathan, "

Schoharie County:
C. K. Frazier, Cobleskill
H. L. Odell, Sharon Springs
John H. Weckel, Breakabeen
C. S. Best, Middleburgh
O. A. Snyder, Schoharie
LeRoy Becker, Cobleskill

Schuyler County:

Seneca County:
James E. Medden, Seneca Falls

Steuben County:
Edward J. Carpenter, Corning
H. B. Nichols, Pultney
Willis S. Cobb, Corning
J. G. Kelly, Hornellsville
John L. Miller, Corning
Thomas H. Pawling, Bath
W. W. Smith, Avoca
C. B. Stephens, Wallace
Stephen M. Switzer, Bradford
James E. Walker, Hornellsville

Suffolk County:
P. Van Benschoten Fowler, Centre Moriches
B. F. Many, Port Jefferson
A. C. Payne, Riverhead
Wm. H. Ross, Brentwood
William B. Savage, East Islip

Sullivan County:

Tioga County:
George M. Cady, Nichols
R. D. Eastman, Berkshire

Tompkins County:
Luzerne Coville, Ithaca

Ulster County:
Daniel Connelly, Kingston
W. D. Hasbrouck, Rondout
E. E. Norwood, Kingston
A. S. Vrooman, High Falls
J. J. Wolf, Rondout
J. L. Preston, Kingston
Buel Latcher, High Falls
Rufus Crawford, West Camp
Jacob Chambers, Kingston
H. C. Keator, Rosendale

Warren County:

Washington County:
George M. Stillman, Argyle
Samuel Pashley, Hartford
D. M. McKenzie, Granville
S. B. Irwin, West Hebron
James T. Park, Sandy Hill

Wayne County:
J. F. Myers, Sodus

Westchester County:
C. F. Chapman, Mt. Kisco
W. F. Greene, Mt. Vernon
Robert T. Howe, Mt. Vernon
C. S. Mead, Portchester
H. B. Brown, Yonkers
C. P. Byington, Croton
W. S. Fleming, Mt. Vernon
E. I. Harrington, Yonkers
R. T. Irvine, Ossining
J. J. Lewin, Port Chester
S. T. Mellen, Poughkeepsie
F. W. Shipman, Mt. Vernon
A. O. Squire, Ossining
G. E. Thomas, Port Chester
L. V. Waldron, Yonkers

Wyoming County:

Yates County:

New York Academy of Medicine:
J. H. Huddleston, 126 West 85th st
Egbert Lefevre, 52 West 56th st
Louis Faugeres Bishop, 30 West 36th st
J. A. Booth, 46 West 49th st
James Ewing, 260 West 57th st

The University and Bellevue Hospital Medical College:

College of Physicians and Surgeons, New York:

N. Y. Post-Graduate Medical School:
Edward Quintard, 145 West 58th st

New York Polyclinic:
R. H. M. Dawbarn, 105 West 74th st

Long Island College Hospital:

Medical Department, Union University:
H. Judson Lipes, Albany

Medical Department, University of Buffalo:
John Parmenter, Buffalo

College of Medicine, Syracuse University:
A. E. Larkin, Syracuse

Rochester Pathological Society:
F. D. Andrew, Rochester

Medical Assoc. of Northern New York:
C. D. Silver, Plattsburgh

Utica Medical Club:
W. R. Marsden, Utica

Utica Medical Library Association:
H. L. Palmer, Utica

Elmira Academy of Medicine:
C. G. R. Jennings, Elmira

Society of Physicians of Canandaigua:
William B. Clapper, Farmington

Practitioners' Society of Rochester:
E. A. Cady-Harris, Rochester

Syracuse Academy of Medicine:
F. H. Flaherty, Syracuse

Amsterdam Medical Society:
J. R. Fairbanks, Amsterdam

Long Island Medical Society:
Wm. S. Hubbard, Brooklyn

Medical Association City of Mt. Vernon and Environs:
Edward S. Newell, Mount Vernon

West End Medical Society of New York City:
Charles G. Kerley, 113 W. 83d st

Gloversville and Johnstown Medical Society:
Arthur C. Hagedorn, Gloversville

The German Medical Society of Brooklyn:
Joseph Meyer, 228 Vernon av

II. Permanent Members.

(By-laws; Chapter I, Sec. 7 to 10; Chapter VII, Sec. 1 to 7. Names in *italics* in the following list are of retired permanent members.)

Year of Election			County
1880	Herman R. Ainsworth,	Addison,	Steuben
1901	Henry A. Alderton,	Brooklyn,	Kings
1887	Charles S. Allen,	Rensselaer,	Rensselaer
1885	Eli Allison,	Wayne,	Steuben
1900	Henry E. Allison,	Fishkill Landing,	Dutchess
1892	Edward B. Angell,	Rochester,	Monroe
1888	Gorham Bacon,	47 West 54th st.,	New York
1858	*Charles G. Bacon* (1884),	Fulton,	Oswego
1902	Charles J. Bacon,	Fulton,	Oswego
1891	Frederick D. Bailey,	Brooklyn,	Kings
1897	Eugene Baker,	Ithaca,	Tompkins
1897	Frank Baldwin,	Brooklyn,	Kings
1893	O. D. Ball,	Albany,	Albany
1895	Eveline P. Ballentine,	Rochester,	Monroe
1889	L. Bolton Bangs,	29 East 46th st.,	New York
1896	Silas J. Banker,	Fort Edward,	Washington
1898	Herbert A. Barney,	Belmont,	Allegany
1896	David A. Barnum,	Cassville,	Oneida
1902	Merritt Wright Barnum,	Ossining,	Westchester
1900	James F. Barker,	Albany,	Albany
1899	Elias H. Bartley,	Brooklyn,	Kings
1899	Joshua L. Barton,	57 East 55th st.,	New York
1884	Milford L. Bates,	Canaan 4 Corners,	Columbia
1876	Eugene Beach,	Gloversville,	Fulton
1895	Albert L. Beahan,	Canandaigua,	Ontario

Year of Election			County
1895	Frank Beebe,	Johnstown,	Fulton
1866	*A. N. Bell* (1881),	Brooklyn,	Kings
1887	Herman Bendell,	Albany,	Albany
1879	M. L. Bennett,	Watkins,	Schuyler
1885	John M. Bigelow,	Albany,	Albany
1897	Chauncey P. Biggs,	Ithaca,	Tompkins
1898	Gilbert Birdsall,	North Brookfield,	Madison
1896	Joseph B. Bissell,	15 West 58th st.,	New York
1901	Louis P. Blair,	McDonough,	Chenango
1896	Herman J. Boldt,	39 East 61st st.,	New York
1896	Reed B. Bontecou,	Troy,	Rensselaer
1888	*C. W. Bourne* (1898),	Hamburgh,	Erie
1888	James P. Boyd,	Albany,	Albany
1900	George A. Bradbury,	Troy,	Rensselaer
1901	W. C. Braislin,	Brooklyn,	Kings
1897	Charles M. Brasted,	Hornellsville,	Steuben
1903	Frederick H. Brewer,	Utica,	Oneida
1874	Elisha H. Bridges,	Ogdensburg,	St. Lawrence
1903	William B. Brinsmade,	Brooklyn,	Kings
1898	Algernon T. Bristow,	Brooklyn,	Kings
1902	George B. Broad,	Syracuse,	Onondaga
1901	Edmond F. Bronk,	Amsterdam,	Montgomery
1894	Charles W. M. Brown,	Elmira,	Chemung
1883	*A. J Browne* (1900),	Utica,	Oneida
1895	William Browning,	Brooklyn,	Kings
1890	Charles E. Bruce,	2 East 127th st.,	New York
1899	Arthur C. Brush,	Brooklyn,	Kings
1888	Edward F. Brush,	Mt. Vernon,	Westchester
1898	George W. Brush,	Brooklyn,	Kings
1897	Joseph D. Bryant,	32 West 48th st.,	New York
1868	*Israel I. Buckbee* (1882),	Fonda,	Montgomery
1887	L. Duncan Bulkley,	531 Madison av.,	New York
1894	Ten Eyck O. Burleson,	Bath,	Steuben
1894	Daniel S. Burr,	Binghamton,	Broome
1889	Stephen Smith Burt,	New York,	New York
1898	William E. Butler,	Brooklyn,	Kings
1894	Glentworth R. Butler,	Brooklyn,	Kings
1892	Peter A. Callan,	35 West 38th st.,	New York
1885	*A. M. Campbell* (1894),	Mt. Vernon,	Westchester
1901	William Francis Campbell	Brooklyn,	Kings
1897	Elon N. Carpenter,	110 West 57th st.,	New York
1884	*H. W. Carpenter* (1895),	Oneida,	Madison
1901	Thomas B. Carpenter,	Buffalo,	Erie
1884	*S. S. Cartwright* (1894),	Roxbury,	Delaware
1890	Charles Carey,	Buffalo,	Erie
1866	*J. E. Casey* (1890),	Mohawk,	Herkimer
1896	Walter Franklin Chappell,	7 East 55th st.,	New York
1879	Walter B. Chase,	Brooklyn,	Kings
1895	William S. Cheeseman,	Auburn,	Cayuga
1886	*Charles H. Chubb* (1899),	Palenville,	Greene
1889	John H. Cipperly,	Troy,	Rensselaer
1895	Frederick L. Classen,	Albany,	Albany
1902	J. M. Clayland,	Brooklyn,	Kings
1895	William T. Clute,	Schenectady,	Schenectady
1894	Henry C. Coe,	8 West 76th st.,	New York
1896	Lewis A. Coffin,	49 West 50th st.,	New York
1895	Carter S. Cole,	122 West 73rd st.,	New York
1891	Philander Collard,	Ossining	Westchester
1899	Herman L. Collyer,	109 East 54th st.,	New York
1867	*Darvin Colvin* (1884),	Clyde,	Wayne

Year of Election			County
1890	William J. Conklin,	Fishkill,	Dutchess
1902	William L. Conklin,	Rochester,	Monroe
1900	Michael P. Conway,	Auburn,	Cayuga
1888	Daniel H. Cook,	Albany,	Albany
1896	James S. Cooley,	Glen Cove,	Queens
1888	J. Leonard Corning,	53 West 38th st.,	New York
1891	Charles N. Cox,	Brooklyn,	Kings
1897	Joseph D. Craig,	Albany,	Albany
1890	*Herman Craft* (1900),	Stone Ridge,	Ulster
1890	Floyd S. Crego,	Buffalo,	Erie
1882	Joseph P. Creveling,	Auburn,	Cayuga
1891	Charles W. Crispell,	Rondout,	Ulster
1898	Montgomery A. Crockett,	Buffalo,	Erie
1899	James K. Crook,	783 Madison av.,	New York
1895	William L. Cuddeback,	Port Jervis,	Orange
1890	Charles M. Culver,	Albany,	Albany
1899	B. Farquhar Curtis,	7 East 41st st.,	New York
1882	Frederic C. Curtis,	Albany,	Albany
1885	John G. Curtis,	327 West 58th st.,	New York
1896	Newton F. Curtis,	White Plains,	Westchester
1896	Alexander Dallas,	New York,	New York
1891	Charles L. Dana,	53 West 53rd st.,	New York
1896	Henry T. Dana,	Cortland,	Cortland
1899	Thomas Darlington,	Kingsbridge av.,	New York
1900	Charles E. Davis,	Albany,	Albany
1902	A. Edward Davis,	42 East 41st st.,	New York
1902	Morris J. Davies,	Utica,	Oneida
1896	Spencer L. Dawes,	Albany,	Albany
1891	Henry H. Deane,	Watertown,	Jefferson
1899	Cornelius W. De Baun,	Fonda,	Montgomery
1893	William B. De Garmo,	56 West 36th st.,	New York
1889	Francis Delafield,	12 West 32d st.,	New York
1894	H. Beekman Delatour,	Brooklyn,	Kings
1902	Edward Bradford Dench,	17 West 46th st.,	New York
1888	Charles A. Dewey,	Rochester,	Monroe
1901	Orson G. Dibble,	Pompey,	Onondaga
1901	Percival F. Dolphin,	Malone,	Franklin
1898	Charles E. Doubleday,	Penn Yan,	Yates
1890	*O. B. Douglas* (1901),	123 East 36th st.,	New York
1899	Henry S. Drayton,	70 Fifth av.,	New York
1878	*H. G. Du Bois* (1900),	Camden,	Oneida
1897	A. Palmer Dudley,	678 Madison av.,	New York
1900	Thomas A. Dundas,	Elmira,	Chemung
1896	Theodore Dunham,	305 West 76th st.,	New York
1900	Z. T. Dunning,	Philmont,	Columbia
1896	Henry Strong Durand,	Rochester,	Monroe
1898	Jesse T. Duryea,	Brooklyn,	Kings
1896	Daniel C. Dye,	Utica,	Oneida
1900	Orlando C. Eastman,	Watertown,	Jefferson
1886	Joseph W. Eddy,	Oswego,	Oswego
1900	R. Jerome Eddy,	Glens Falls,	Warren
1895	George M. Edebohls,	59 West 49th st.,	New York
1893	John Edwards,	Gloversville,	Fulton
1870	*Ellsworth Elliot* (1887),	48 West 36th st.,	New York
1896	Elmore E. Elliott,	Catskill,	Greene
1892	Henry L. Elsner,	Syracuse,	Onondaga
1875	William S. Ely,	Rochester,	Monroe
1892	Z. Taylor Emery,	Brooklyn,	Kings
1902	Bache McEvers Emmet,	18 East 30th st.,	New York
1871	*Thos. A. Emmet* (1902),	91 Madison av.,	New York
1891	George A. Evans,	Brooklyn,	Kings

Year of Election			County
1898	Henry A. Fairbairn,	Brooklyn,	Kings
1889	Jas. D. Featherstonhaugh	Cohoes,	Albany
1892	Mahlon Felter,	Troy,	Rensselaer
1900	Charles G. J. Finn,	Hempstead,	Queens
1890	Edward D. Fisher,	19 West 52d st.,	New York
1896	Arthur Lyman Fisk,	41 West 50th st.,	New York
1888	Henry Flood,	Elmira,	Chemung
1899	Henry Hall Forbes,	96 Park av.,	New York
1888	Willis E. Ford,	Utica,	Oneida
1896	John Addison Fordyce,	66 Park av.,	New York
1903	George Bingham Fowler,	18 East 58 th st.,	New York
1889	George R. Fowler,	Brooklyn,	Kings
1882	George Henry Fox,	18 East 31st st.,	New York
1902	Rowland G. Freeman,	205 West 57th st.,	New York
1862	*S. H. Freeman* (1886),	Albany,	Albany
1891	Crawford E. Fritts,	Hudson,	Columbia
1897	Henry P. Frost,	Buffalo,	Erie
1899	Jacob Fuhs,	Brooklyn,	Kings
1893	Earl D. Fuller,	Utica,	Oneida
1902	Henry L. Furbeck,	St. Johnsville,	Montgomery
1897	William D. Garlock,	Little Falls,	Herkimer
1902	Henry A. Gates,	Delhi,	Delaware
1885	John Gerin,	Auburn,	Cayuga
1887	Arpad G. Gerster,	34 East 75th st.,	New York
1884	Virgil P. Gibney,	16 Park av.,	New York
1899	Charles Langdon Gibson,	48 West 49th st.,	New York
1893	William M. Gibson,	Utica,	Oneida
1902	Herbert F. Gillette	Cuba,	Allegany
1889	James H. Glass,	Utica,	Oneida
1891	De Witt A. Gleason,	Oxford,	Chenango
1901	W. Stanton Gleason,	Newburg,	Orange
1900	Charles H. Glidden,	Little Falls,	Herkimer
1892	D. H. Goodwillie,	New York,	New York
1896	Hermon C. Gordinier,	Troy,	Rensselaer
1897	Egbert H. Grandin,	36 East 58th st.,	New York
1894	George Graves,	Herkimer,	Herkimer
1881	Henry Gray,	Greenwich,	Washington
1901	Emil Gruening,	36 East 57th st.,	New York
1902	Ramon Guiteras,	75 West 55th st.,	New York
1888	Alexander Hadden,	155 East 51st st.,	New York
1887	William Hailes,	Albany,	Albany
1897	Lorenzo Hale,	Albany,	Albany
1897	Orlando J. Hallenbeck,	Canandaigua,	Ontario
1898	Thomas H. Halsted,	Syracuse,	Onondaga
1903	William B. Hanbidge,	Ogdensburgh,	St. Lawrence
1903	William Henry Happel,	Albany,	Albany
1892	John B. Harvie,	Troy,	Rensselaer
1899	Hickson F. Hart,	Shrub Oak,	Westchester
1895	Josiah Hasbrouck,	Port Ewen,	Ulster
1899	Wm. Henry Haskin,	New York	New York
1894	Herman E. Hayd,	Buffalo,	Erie
1903	William E. Hayes,	Frankfort,	Herkimer
1889	John L. Heffron,	Syracuse,	Onondaga
1898	Alfred W. Henckell,	Rochester,	Monroe
1889	Edgar M. Hermance,	Yonkers,	Westchester
1894	Wallace J. Herriman,	Rochester,	Monroe
1900	Francis W. Higgins,	Cortland,	Cortland
1885	Henry R. Hopkins,	Buffalo,	Erie
1898	D. Walker Houston,	Troy,	Rensselaer
1874	*Bleecker L. Hovey* (1885)	Rochester,	Monroe
1891	Eugene H. Howard,	Rochester,	Monroe

Year of Election			County
1892	Wm. R. Howard,	Rochester,	Monroe
1886	Lucien Howe,	Buffalo,	Erie
1891	John T. Howell,	Newburgh,	Orange
1896	Joseph B. Hullett,	Middletown,	Orange
1893	Henry Hun,	Albany,	Albany
1903	James Zalmon Hunt,	Lowville,	Lewis
1899	Dwight W. Hunter,	80 West 40th st.,	New York
1897	George Huntington,	La Grangeville,	Dutchess
1901	Arthur W. Hurd,	Buffalo,	Erie
1894	Joel W. Hyde,	Brooklyn,	Kings
1903	Edward Marshall Hyland,	Utica,	Oneida
1893	George T. Jackson	15 East 29th st.,	New York
1875	Abraham Jacobi,	19 East 47th st.,	New York
1892	Nathan Jacobson,	Syracuse,	Onondaga
1893	Walter W. Jamieson,	Syracuse,	Onondaga
1895	Reuben Jeffery,	Norwich,	Chenango
1886	Charles Jewett,	Brooklyn,	Kings
1897	Frederick A. Jewett,	Brooklyn,	Kings
1901	John H. Jewett,	Canandaigua,	Ontario
1899	Henrietta P. Johnston,	206 East 15th st.,	New York
1899	George P. Johnson,	Mexico,	Oswego
1882	Wm. H. Johnston,	Port Leydon,	Lewis
1903	Thomas Z. Jones,	Waterville,	Oneida
1900	William B. Jones,	Rochester,	Monroe
1900	Andrew V. Jova,	Newburgh,	Orange
1902	James E. Kelly,	117 East 59th st.,	New York
1903	W. Clinton Kellogg,	Syracuse,	Onondaga
1896	Judson G. Kilbourn,	Utica,	Oneida
1903	Jack Killen,	Binghamton,	Broome
1901	James W. King,	Stottville,	Columbia
1891	Osman F. Kinloch,	Troy,	Rensselaer
1899	Cyrile P. Kirley,	Lowville,	Lewis
1880	Herman Knapp,	26 West 40th st.,	New York
1901	James W. Knapp,	Canastota,	Madison
1898	William C. Krauss,	Buffalo,	Erie
1895	Austin La Monte,	Carmel,	Putnam
1898	Wilbur F. Lamont,	Catskill,	Greene
1896	Louis Nott Lanehart,	Hempstead,	Queens
1890	Charles M. Lefler,	Gloversville,	Fulton
1890	Maurice J. Lewi,	31 West 84th st.,	New York
1884	Daniel Lewis,	252 Madison av.,	New York
1902	Robert Lewis, Jr.,	48 West 40th st.,	New York
1892	Samuel Lloyd,	12 West 50th st.,	New York
1892	Eli H. Long,	Buffalo,	Erie
1901	Earl P. Lothrop,	Buffalo,	Erie
1882	E. H. Loughran,	Kingston,	Ulster
1899	Bradford C. Loveland,	Syracuse,	Onondaga
1871	Frank S. Low,	Pulaski,	Oswego
1898	David S. Lucas,	Brooklyn,	Kings
1902	William V. V. Mabon,	Ogdensburg,	St. Lawrence
1891	Carlos F. MacDonald,	85 Madison av.,	New York
1902	Alexander E. Macdonald	Manhat. State Hosp.,	New York
1895	Willis G. Macdonald,	Albany,	Albany
1896	John C. MacEvitt,	Brooklyn,	Kings
1900	Andrew MacFarlane,	Albany,	Albany
1894	Frank Madden,	Plattsburgh,	Clinton
1890	William Maddren,	Brooklyn,	Kings
1893	A. H. Mambert,	Rondout,	Ulster
1901	Frederick A. Mandeville,	Rochester,	Monroe
1895	John Mann,	Jericho,	Queens

Year of Election			County
1897	Matthew D. Mann,	Buffalo,	Erie
1902	Francis H. Markoe,	15 East 49th st.,	New York
1891	Frank W. Marlow,	Syracuse,	Onondaga
1903	Thomas Masson,	Cape Vincent,	Jefferson
1889	A. Ross Matheson,	Brooklyn,	Kings
1879	Arthur Mathewson,	Brooklyn,	Kings
1899	Sands C. Maxson,	Utica,	Oneida
1901	E. S. McClellan,	Saranac Lake,	Franklin
1902	Roderick C. McClennan,	Syracuse,	Onondaga
1894	George M. McCombs,	Frankfort,	Herkimer
1894	John A. McCorkle,	Brooklyn,	Kings
1902	James F. McKernon,	62 West 52d st.,	New York
1903	Sylvester J. McNamara,	Brooklyn,	Kings
1895	George McNaughton,	Brooklyn,	Kings
1900	Edward Meany,	Ithaca,	Tompkins
1884	*Alfred Mercer* (1899),	Syracuse,	Onondaga
1898	A. Clifford Mercer,	Syracuse,	Onondaga
1897	Henry E. Mereness,	Albany,	Albany
1900	Edwin H. Merriam,	Coxsackie,	Greene
1888	Cyrus S. Merrill,	Albany,	Albany
1897	George V. R. Merrill,	Elmira,	Chemung
1892	Willy Meyer,	700 Madison av.,	New York
1895	Aaron B. Miller,	Syracuse,	Onondaga
1888	Lewis H. Miller,	Brooklyn,	Kings
1892	Theodore D. Mills,	Middletown,	Orange
1903	James H. Mitchell,	Cohoes,	Albany
1886	Wm. F. Mittendorf,	140 Madison av.,	New York
1891	Joseph Moffatt,	Washingtonville,	Orange
1896	Edward L. Mooney,	Syracuse,	Onondaga
1896	William A. Moore,	Binghamton,	Broome
1896	John Moroney,	Elmira,	Chemung
1892	Robert T. Morris,	58 West 56th st.,	New York
1898	Robert J. Morrison,	Brooklyn,	Kings
1895	Samuel R. Morrow,	Albany,	Albany
1896	William James Morton,	19 East 28th st.,	New York
1896	J. Montgomery Mosher,	Albany,	Albany
1894	Edward W. Mulligan,	Rochester,	Monroe
1903	George Stephen Munson,	Albany,	Albany
1902	Dwight H. Murray,	Syracuse,	Onondaga
1899	Grace Peckham Murray,	661 Fifth av.,	New York
1896	T. Halsted Myers,	54 West 50th st.,	New York
1898	Herman V. Mynderse,	Schenectady,	Schenectady
1898	Charles D. Napier,	Brooklyn,	Kings
1902	Lewis Knode Neff,	1213 Park av.,	New York
1893	William J. Nellis,	Albany,	Albany
1903	Leo H. Neuman,	Albany,	Albany
1892	Albert S. Newcomb,	206 West 71st st.,	New York
1881	C. E. Nichols,	Troy,	Rensselaer
1902	Henry S. Norris,	10 West 49th st.,	New York
1895	William P. Northrup,	57 East 79th st.,	New York
1890	George H. Oliver,	Malone,	Franklin
1903	Daisy M. Orleman,	Peekskill,	Westchester
1890	May R. Owen,	Brooklyn,	Kings
1893	Albert H. Palmer,	Marlborough,	Ulster
1892	Roswell Park,	Buffalo,	Erie
1897	Frederick H. Parker,	Auburn,	Cayuga
1895	Clair S. Parkhill,	Hornellsville,	Steuben
1863	*W.H.H.Parkhurst* (1883)	Frankfort,	Herkimer
1902	John Parmenter,	Buffalo,	Erie
1887	Edward L. Partridge,	19 Fifth av.,	New York

Year of Election			County
1893	O. W. Peck,	Oneonta,	Otsego
1899	Fayette H. Peck,	Utica,	Oneida
1881	*Solomon H. Peck* (1892),	Ithaca,	Tompkins
1902	James Pedersen,	20 East 46th st.,	New York
1893	T. Kirkland Perry,	Albany,	Albany
1891	Wendell C. Phillips,	40 West 47th st.,	New York
1891	Lewis S. Pilcher,	Brooklyn,	Kings
1903	Charles Winfield Pilgrim,	Poughkeepsie,	Dutchess
1894	Milton G. Planck,	Schenectady,	Schenectady
1896	Warren O. Plimpton,	19 East 28th st.,	New York
1897	John Osborne Polak,	Brooklyn,	Kings
1893	George P. K. Pomeroy,	Stuyvesant,	Columbia
1881	*Thomas R. Pooley* (1903),	107 Madison av.,	New York
1869	*Charles H. Porter* (1900),	Albany,	Albany
1903	Ezra B. Potter,	Rochester,	Monroe
1897	Marion Craig Potter,	Rochester,	Monroe
1883	Wm. Warren Potter,	Buffalo,	Erie
1893	J. Wilson Poucher,	Poughkeepsie,	Dutchess
1892	Seneca D. Powell,	12 West 40th st.,	New York
1879	*Jonathan S. Prout* (1897),	Brooklyn,	Kings
1897	John H. Pryor,	Buffalo,	Erie
1877	Alfred E. M. Purdy,	304 Madison av.,	New York
1899	Barna E. Radeker,	Deposit,	Broome
1896	Julius B. Ransom,	Dannemora,	Clinton
1900	James E. Reed,	Schenectady,	Schenectady
1894	Charles M. Rexford,	Watertown,	Jefferson
1889	Clarence C. Rice,	123 East 19th st.,	New York
1900	Nelson G. Richmond,	Fredonia,	Chautauqua
1896	DeWitt C. Rodenhurst,	Philadelphia,	Jefferson
1882	John O. Roe,	Rochester,	Monroe
1878	D. B. St. John Roosa,	20 East 30th st.,	New York
1900	Arthur G. Root,	Albany,	Albany
1898	Lewis W. Rose,	Rochester,	Monroe
1898	George W. Rossman,	Ancram,	Columbia
1900	James E. Sadlier,	Poughkeepsie,	Dutchess
1891	Thomas E. Satterthwaite	7 East 80th st.,	New York
1899	Reginald H. Sayre,	9 East 45th st.,	New York
1901	Peter Scott,	Brooklyn,	Kings
1903	Thomas P. Scully,	Rome,	Oneida
1903	Frank W. Sears,	Binghamton,	Broome
1897	Frederick W. Sears,	Syracuse,	Onondaga
1901	Frank M. Severson,	Seneca Falls,	Seneca
1885	*George Seymour* (1900),	Utica,	Oneida
1893	William W. Seymour,	Troy,	Rensselaer
1897	Friend W. Shaffer,	Gloversville,	Fulton
1898	John E. Sheppard,	Brooklyn,	Kings
1884	Samuel Sherwell,	Brooklyn,	Kings
1901	Frederic J. Shoop,	Brooklyn,	Kings
1870	George F. Shrady,	8 East 66th st.,	New York
1902	William Simmons,	Brooklyn,	Kings
1898	William H. Skene,	Brooklyn,	Kings
1900	Erasmus D. Skinner,	Mineola,	Queens
1897	Frederick W. Slocum,	Camillus,	Onondaga
1899	Charles E. Smith,	Whitesboro,	Oneida
1903	Eugene Alfred Smith,	Buffalo,	Erie
1893	H. Lyle Smith,	Hudson,	Columbia
1900	Nathan Smith,	Richmondville,	Schoharie
1902	Sargent F. Snow,	Syracuse,	Onondaga
1886	James D. Spencer,	Watertown,	Jefferson
1892	Henry S. Stearns,	45 West 58th st.,	New York

Year of Election			County
1890	Byron U. Steenberg,	Albany,	Albany
1902	F. H. Stephenson,	Syracuse,	Onondaga
1900	Alexander A. Stern,	Kingston,	Ulster
1892	William Stevens,	70 West 52nd st.,	New York
1879	Charles L. Stiles,	Owego,	Tioga
1901	David V. Still,	Johnstown,	Fulton
1897	William O. Stillman,	Albany,	Albany
1880	Enoch V. Stoddard,	Rochester,	Monroe
1889	Charles Stover,	Amsterdam,	Montgomery
1882	Frederic R. Sturgis,	16 West 32nd st.,	New York
1885	A. Walter Suiter,	Herkimer,	Herkimer
1892	Peter L. Suits,	Tribe's Hill,	Montgomery
1896	Leander Swartwout,	Prospect,	Oneida
1896	T. Oliver Tait,	Rochester,	Monroe
1900	John H. Taylor,	Holley,	Orleans
1898	John Warren Thorp,	Oxford,	Chenango
1897	C. F. Timmerman,	Amsterdam,	Montgomery
1900	William A. Tomes,	Brooklyn,	Kings
1888	David M. Totman,	Syracuse,	Onondaga
1897	C. Wilmot Townsend,	New Brighton,	Richmond
1896	Wisner R. Townsend,	125 West 58th st.,	New York
1902	C. Howard Travell,	Troy,	Rensselaer
1889	Willis G. Tucker,	Albany,	Albany
1880	Robert K. Tuthill,	Poughkeepsie,	Dutchess
1892	Francis Valk,	164 East 61st st.,	New York
1895	Joshua M. Van Cott,	Brooklyn,	Kings
1902	John Vanderpoel,	36 West 39th st.,	New York
1889	S. O. Vanderpoel,	47 East 25th st.,	New York
1899	Waldron B. Vanderpoel,	106 East 24th st.,	New York
1876	Albert Vander Veer	Albany,	Albany
1902	Ely Van de Warker,	Syracuse,	Onondaga
1901	Charles M. Van Dyke,	Himrod,	Yates
1899	Frank Van Fleet,	60 East 77th st.,	New York
1900	Howard Van Rensselaer,	Albany,	Albany
1886	Eugene Van Slyke,	Albany,	Albany
1898	Major A. Veeder,	Lyons,	Wayne
1890	Waterman A. Vincent,	Three-Mile Bay,	Jefferson
1899	Charles G. Wagner,	Binghamton,	Broome
1892	Ralph Waldo,	60 West 50th st.,	New York
1895	John J. Walsh,	Buffalo,	Erie
1901	James P. Warbasse,	Brooklyn,	Kings
1896	David B. Ward,	Poughkeepsie,	Dutchess
1873	R. Halstead Ward,	Troy,	Rensselaer
1883	Samuel B. Ward	Albany,	Albany
1885	*John S. Warren* (1898),	150 West 48th st.,	New York
1883	David Webster,	327 Madison av.,	New York
1901	William S. Webster,	Liberty,	Sullivan
1898	George C. Weiss,	Mt. Vernon,	Westchester
1895	Ernest Wende,	Buffalo,	Erie
1901	Grover W. Wende,	Buffalo,	Erie
1896	George T. Wetmore,	36 West 35th st.,	New York
1899	William E. Wetmore,	Utica,	Oneida
1894	Hamilton D. Wey,	Elmira,	Chemung
1889	John T. Wheeler,	Chatham,	Columbia
1883	John W. Whitbeck,	Rochester,	Monroe
1902	Frederick Whiting,	47 West 50th st.,	New York
1895	Reynold W. Wilcox,	679 Madison av.,	New York
1898	Alfred G. Wilding,	Malone,	Franklin
1892	Robert G. Wilding.	Malone,	Franklin
1883	Charles E. Willard,	Catskill,	Greene

Year of Election			County
1898	Edward S. Willard,	Watertown,	Jefferson
1895	George O. Williams,	Greene,	Chenango
1893	Henry T. Williams,	Rochester,	Monroe
1901	Herbert U. Williams,	Buffalo,	Erie
1895	James McF. Winfield,	Brooklyn,	Kings
1881	Charles E. Witbeck,	Cohoes,	Albany
1892	George H. Witter,	Wellsville,	Allegany
1889	Rudolph A. Witthaus,	45 West 32nd st.,	New York
1903	Wilbur D. Wolff,	Rochester,	Monroe
1900	George H. Wood	Antwerp,	Jefferson
1895	J. Walter Wood,	Port Richmond,	Richmond
1897	W. Gill Wylie,	28 West 40th st.,	New York
1901	Edward J. Wynkoop,	Syracuse,	Onondaga
1900	Charles D. Young,	Rochester,	Monroe
1903	William Henry Zabriskie,	Glen Cove,	Queens
1895	Frederick W. Zimmer,	Rochester,	Monroe

III. Honorary Members

Year of Election

1889 Francis Bacon, New Haven, Connecticut
1888 B. F. Baer, Philadelphia, Pa.
1882 R. Bartholow, Philadelphia, Pa.
1880 J. S. Billings, Philadelphia, Pa.
1877 W. A. F. Brown, Dumphries, Scotland
1886 E. N. Brush, Baltimore, Md.
1881 J. C. Bucknill, London, Eng.
1894 Arthur T. Cabot, Boston, Mass.
1903 R. H. Chittenden, New Haven, Ct.
1885 T. S. Clouston, Edinburgh, Scotland
1866 N. S. Davis, Chicago, Ill.
1900 John B. Deaver, Philadelphia, Pa.
1903 Francis X. Dercum, Philadelphia, Pa.
1875 F. Dichiara, Palermo, Italy
1890 George J. Engelmann, Boston, Mass.
1875 E. H. Gregory, St. Louis, Mo.
1887 A. P. Grinnell, Burlington, Vt.
1886 T. Hansen, Copenhagen, Denmark
1889 Reginald Harrison, Liverpool, England
1879 C. Heath, London, England
1894 O. E. Herrick, Grand Rapids, Michigan
1882 C. N. Hewitt, Red Wing, Minn.
1891 W. H. Hingston, Montreal, Canada
1877 W. S. Hopkins, Vergennes, Vt.
1891 J. Nevins Hyde, Chicago, Ill.
1870 Ralph Isham, Chicago, Ill.
1899 William W. Keen, Philadelphia, Pa.
1887 E. Landolt, Paris, France
1873 H. C. Lombard, Geneva, Switzerland

Year of Election

1871 W. McCollum, Brooklyn, N. Y.
1897 J. Lee McComas, Oakland, Md.
1892 Lewis S. McMurtry, Louisville, Kentucky
1872 R. McNamara, Dublin, Ireland
1889 Howard Marsh, London, Eng.
1898 Joseph M. Matthews, Louisville, Ky.
1879 L. A. Mercier, Paris, France
1877 S. Weir Mitchell, Philadelphia, Pa.
1891 E. E. Montgomery, Philadelphia, Pa.
1899 William Osler, Baltimore, Md.
1894 J. H. Packard, Philadelphia. Pa.
1891 Joseph Price, Philadelphia, Pa.
1892 Chas. A. L. Reed, Cincinnati, Ohio
1899 Maurice H. Richardson, Boston, Mass.
1902 B. Merrill Ricketts, Cincinnati, Ohio
1882 Sidney Ringer, London, Eng.
1883 C. L. Robertson, London, Eng.
1892 Jas. F. W. Ross, Toronto, Can.
1889 Max Schede, Hamburg, Germany
1888 F. J. Shepherd, Montreal, Can.
1894 George M. Sternberg, Washington, D. C.
1873 R. Stokes, Dublin, Ireland
1894 I. S. Stone, Washington, D. C.
1881 W. H. Taylor, Cincinnati, O.
1873 T. J. Turner, U. S. Navy
1902 J. Collins Warren, Boston, Mass.
1877 L. de Wecker, Paris, France
1891 H. C. Wood, Philadelphia, Pa.
1892 A. H. Wright, Toronto, Can.

COUNTY MEDICAL SOCIETIES

MEDICAL SOCIETY OF THE COUNTY OF ALBANY.

(Organized in 1806.)

MEETINGS.—Annual, second Tuesday in May; semi-annual, second Tuesday in October. Stated meetings are held Wednesday evening, at Alumni Hall, Albany Medical College, at least once a month, from October to May.

Officers. (May, 1903.)

Cyrus S. Merrill, *President.* John Archibold, *Vice-President.*
Henry L. K. Shaw, *Secretary.* William H. George, *Treasurer.*
Censors: J. M. Mosher, A. Vander Veer, W. G. Macdonald, A. H. Traver, B. U. Steenberg.

Members.

(Where the town is not mentioned Albany is to be understood.)

Ira Applebee, 318 S. Pearl st
J. L. Archambault, Cohoes
La Salle Archambault, Cohoes
John Archibold, Cohoes
Robert Babcock, 102 Lancaster st
Theodore P. Bailey, 95 Eagle st
O. D. Ball, 691 Broadway
James F. Barker, 54 Clinton av
E. A. Bartlett, 20 S. Hawk st
E. J. Bedell, Becker's Corners
Edward Bell, Cohoes
Herman Bendell, 178 State st
J. M. Bigelow, 54 Eagle st
Anne T. Bingham, 82 S. Swan st
L. E. Blair, 204 State st
Joseph H. Blatner, 132 Hudson av
A. J. Blessing, 219 State st
Geo. Blumer, Bender Hygienic Lab.
James P. Boyd, 152 Washington av
W. S. Bristol, 76 S. Ferry st
A. S. Capron, 78 Hudson av
T. L. Carroll, 297 Lark st
Daniel C. Case, Slingerlands
F. L. Classen, 34 Trinity place
E. V. Colbert, 62 Hudson av
D. H. Cook, 264 Clinton av
J. L. Cooper, 86 Clinton av
Joseph D. Craig, 12 Ten Broeck st
Frederic Crounse, Altamont
C. M. Culver, 36 Eagle st
F. C. Curtis, 17 Washington av
F. C. E. Daunais, Cohoes
C. E. Davis, 15 Washington av
S. L. Dawes, 255 State st
M. J. Dwyer, 268 Hamilton st
Noah L. Eastman, 427 Clinton av
A. W. Elting, 247 State st
J. D. Featherstonhaugh, Cohoes
F. H. Fisk, 1 Clinton square
S. H. Freeman, 77 Columbia st
G. H. Furbeck, Cohoes
A. L. George, 769 Broadway
Wm. H. George, 318 Central av
John Giffen, 82 Philip st
W. I. Goeway, 225 Hamilton st
F. R. Greene, 542 Central av
Gerald Griffith, 114 Grand st
F. N. Guyer, 181 Clinton av
Wm. Hailes, 197 Hamilton st
Lorenzo Hale, 50 Clinton av
W. S. Hale, 50 Clinton av
W. H. Happel, 351 S. Pearl st
J. U. Haines, Cohoes
John V. Hennessy, 35 Clinton av
E. E. Hinman, 202 Lark st
Alfred B. Huested, State c. Eagle st
Henry Hun, 149 Washington av
T. W. Jenkins, 246 Hamilton st
F. M. Joslin, Voorheesville
P. J. Keegan, 48 Clinton av
J. A. Lanahan, 31 Jefferson st
M. S. Leavy, 9 Watervliet av
Louis Le Brun, 71 S. Ferry st
G. G. Lempe, 42 Eagle st
W. G. Lewi, 296 Lark st
H. Judson Lipes, 178 Washington av
G. E. Lochner, 196 State st
Howard E. Lomax, 114 Jay st
W. G. Macdonald, 27 Eagle st
A. Macfarlane, 198 Washington av
M. MacHarg, 806 Madison av
C. C. McCulloch, 363 S. Pearl st
J. F. McGarrahan, Cohoes
E. N. K. Mears, 1 Washington av
H. E. Mereness, 184 State st
C. S. Merrill, 21 Washington av
J. H. Mitchell, Cohoes
J. D. Montmarquet, Cohoes
C. H. Moore, 21 Washington av
J. M. Moore, 375 Madison av
Samuel R. Morrow, 29 S. Hawk st
J. M. Mosher, 170 Washington av
G. T. Moston, 611 Central av
George S. Munson, 30 Eagle st
W. H. Murray, 292 Lark st
C. L. Myers, 232 N. Pearl st

W. J. Nellis, 210 State st
L. H. Neuman, 194 State st
D. V. O'Leary, 10 Ash Grove place
G. W. Papen, 268 Madison av
H. D. Pease, 116 Washington av
G. Upton Peltier, Cohoes
T. K. Perry, 174 Second st
Alonzo F. Powell, Coeymans
W. L. Purple, 46 Second st
M. J. Read, Indian Fields
Martin Reid, Reidsville
C. H. Richardson, 217 State st
James F. Rooney, 123 Grand st
Arthur G. Root, 218 State st
W. B. Rossman, 324 Hamilton st
Thomas A. Ryan, 47 Eagle st
W. B. Sabin, Watervliet
F. Saurbrie, Clarksville
Arthur Sautter, 220 State st
Wm. L. Schutter, 279 Clinton av
Seth G. Shanks, 547 Clinton av
H. L. K. Shaw, 198 Washington av
Ralph Sheldon, 291 State st
J. H. Skillicorn, 271 Hudson av
C. H. Smith, 246 Washington av
J. E. Smith, 246 Washington av
B. U. Steenberg, 1 Ten Broeck st
M. D. Stevenson, 51 S. Ferry st
W. O. Stillman, 27 State st
Elizabeth Sweet, 13 Ten Broeck st
Mary Sweet, 82 S. Swan st
R. H. Tedford, Jr., 116 Central av
C. F. Theisen, 172 Washington av
A. H. Traver, 809 Madison av
T. Markley Trego, 33 Clinton av
Benno G. Troidle, 115 Central av
Willis G. Tucker, College Building
G. L. Ullman, 92 Central av
L. Van Auken, Watervliet
Albert Vander Veer, 28 Eagle st
Edgar A. Vander Veer, 28 Eagle st
Howard Van Rensselaer, 123 State st
E. Van Slyke, 139 Madison av
W. J. Wansboro, 113 Lark st
S. B. Ward, 281 State st
J. B. Washburne, Delmar
James W. Wiltse, 6 S. Hawk st
L. B. Winne, 72 Livingston av
C. E. Witbeck, Cohoes
Harriet A. Woodward, 42 Chestnut st

Number of Members, 135.

NOTE.—For Delegates from County Societies see *Delegates*, preceding.

Deceased: Theodore W. Nellis, Albany, April 14, 1902; John Thompson, Albany, July 20, 1902, æt. 65; T. F. C. Van Allen, Albany, October 28, 1902, æt. 41; Adam T. Van Vranken, Watervliet, January 19, 1903, æt. 52.

MEDICAL SOCIETY OF THE COUNTY OF ALLEGANY.

(Date of organization unknown.)

MEETINGS.—Annual, second Wednesday in May; quarterly, second Wednesday in August, November, and February.

Officers. (1903.)

H. L. Hulett, *President.* J. C. Young, *Vice-President.*
George E. Burdick, *Secretary and Treasurer.*
Censors: George E. Burdick, J. W. Collar, H. A. Barney, G. H. Witter, J. C. Young.

Members.

Otis Allen, Cuba
E. W. Ayers, Richburg
F. C. Ballard, Rushford
H. A. Barney, Belmont
George E. Burdick, Andover
O. E. Burdick, Little Genesee
J. W. Collar, Wellsville
F. E. Comstock, Wellsville
W. O. Congdon, Cuba
H. E. Cooley, Angelica
Dorr Cutler, Bolivar
C. C. Deming, Friendship
N. H. Fuller, Friendship
E. C. Gilbert, Angelica
H. F. Gillette, Cuba
F. E. Howard, Friendship
H. L. Hulett, Allentown
Fred. T. Koyle, Wellsville
O. N. Latham, Bolivar
Elbert W. La Wall, Scio
Wm. Litchfield, Cuba
W. H. Loughead, Nile
C. M. Post, Alfred
A. J. Remington, Whitesville
George W. Roos, Wellsville
C. O. Sayers, Boliver
Mark Sheppard, Alfred
C. M. Stewart, Hume
Edith Stewart, Hume
T. S. Thomas, Cuba
F. H. Van Orsdale, Belmont
B. Williamson, Friendship
G. H. Witter, Wellsville
J. C. Young, Cuba

Number of Members, 34.

Deceased: R. V. Crittenden, Friendship; John P. Crosby, Belmont; H. P. Saunders, Alfred.

MEDICAL SOCIETY OF THE COUNTY OF BROOME.

(Organized July 4, 1806.)

MEETINGS.—Annual, first Tuesday in October; semi-annual, first Tuesday in April.

Officers. (October, 1902.)

W. S. Overton, *President.* C. R. Seymour, *Vice-President.*
Jack Killen, *Secretary.* E. H. Wells, *Treasurer.*
Censors: J. G. Orton, J. H. Chittenden, J. M. Farrington, R. A. Seymour, A. F. Taylor.

Members.

(Where the name of the town is not given Binghamton is to be understood.)

S. P. Allen, Lestershire
J. D. Appley, Harpersville
W. E. Ard, 87 Front st
S. Taylor Barton, Lestershire
H. F. Beardsley, 500 Chenango st
Ray Beardsley, 502 Chenango st
J. M. Booth, West Colesville
D. S Burr, 145 Court st
Charles Butler, Nineveh
J. H. Chittenden, 51 Main st
E. N. Christopher, Union
W. D. Christopher, Union
J. C. Comstock, 1 Lewis st
W. W. Clark, Maine
R. R. Daly, 234 Washington st
E. F. Day, 96 Court st
Dwight Dudley, Maine
I. C. Edson, Windsor
H. O. Ely, 62 Front st
J. M. Farrington, 11 Jay st
L. D. Farnham, 42 Main st
F. L. Forker, 38 Fayette st
K. C. French, Lisle
C. W. Green, Jay st
J. D. Guy, Chenango Forks
W. D. Hardy, Union
L. H. Hills, 3 Dwight Block
I. A. Hix, Carroll st
F. P. Hough, 89½ Oak st
H. S. Hutchinson, Grand Boulevard
R. C. Irving, Main st
J. W. Jansen, 28 Exchange st
J. Killen, 74 Front st
Frank MacKnight, Exchange st
J. H. Martin, 172 Court st
F. M. Miller, 145 Court st
W. A. Moore, 223 Washington st
Mary O'Malley, State Hospital
D. C. O'Neil, 226 Front st
W. S. Overton, 114 Court st
J. G. Orton, Henry st and Prospect av
Ransom J. Perry, Whitney Point
J. F. Pratt, 115 Front st
F. W. Putnam, 210 Vestal av
John F. Place, Jr., 134 Court st
L. H. Quackenbush, 26 Arthur st
B. E. Radeker, Deposit
C. B. Richards, 86 Front st
Charles Roberts, Chenango st
Flora Ruch, Lestershire
F. W. Sears, cor. Front and Perry sts
Charles R. Seymour, 258 Court st
R. A. Seymour, Whitney Point
Edward L. Smith, 60 Carroll st
George B. Stanwix, 262 Washington st
A. P. Summers, State Hospital
A. F. Taylor, Castle Creek
E. L. Teed, Centre Lisle
J. L. Van Alstyne, 100 Hawley st
T. B. Van Alstyne, 324 Chenango st
Charles G. Wagner, State Hospital
E. H. Wells, 14 Congdon Place
W. A. White, State Hospital
J. S. Whitney, Union
W. W. Whitney, Union
W. H. Wilson, Lestershire
L. D. Witherill, Union.

Number of Members, 67.

MEDICAL SOCIETY OF THE COUNTY OF CATTARAUGUS.

MEETINGS.—Annual, at Salamanca, the first Tuesday in May; quarterly, on the first Tuesday in August, November, and February.

Officers. (May, 1903.)

G. W. Winterstine, *President.* J. Sutherland Wright, *Vice-President.*
J. A. Taggert, *Secretary and Treasurer.*
Censors: A. D. Lake, F. C. Beals, G. W. Winterstine.

Members.

J. R. Allen, Olean
H. J. Ashley, Machias
Fred C. Beals, Salamanca
S. S. Bedient, Little Valley
F. P. Blair, Allegany
J. P. Booth, Olean

A. L. Borden, Gowanda
I. H. Boyd, Olean
E. R. Burdick, Olean
A. K. Corbin, Limestone
E. M. Coss, Cattaraugus
Salina P. Colegrove, Salamanca
W. C. Dallanbaugh, Olean
Lyman L. Deck, Salamanca
Myron E. Fisher, Delavan
M. C. Follett, Olean
William Follett, Sandusky
M. C. Hawley, East Randolph
Jacob S. Hicks, Allegany
Wm. B. Johnston, Ellicottville
William W. Jones, Dayton
Charles Kelley, Franklinville
Clarence King, Machias
Charles P. Knowles, Olean
Frederick Krehbiel, Delevan
A. D. Lake, Gowanda
T. B. Loughlin, Olean
John McCartey, Limestone
George McIntosh, Cattaraugus
S. B. McClure, Allegany
Wm. E. McDuffie, Olean
O. S. Martin, Salamanca
J. E. K. Morris, Olean
William H. Mountain, Olean
Charles L. Randall, Franklinville
W. B. Reynolds, Olean
J. H. Sackrider, East Randolph
E. M. Shaffner, Great Valley
Alfred Smallman, Ellicottville
Cassar Smith, Olean
H. D. Snover, Steamburgh
James A. Taggert, Salamanca
William Tefft, S. Dayton
O. A. Tompkins, Randolph
Edward Torrey, Allegany
W. H. Vincent, Hinsdale
H. D. Walker, Franklinville
C. M. Walrath, Ellicottville
G. W. Winterstine, Portville
E. D. Williams, East Otto
J. Sutherland Wright, Little Valley

Number of Members, 51.

Deceased: John L. Eddy, Olean, March 30, 1902, æt. 73; A. C. Porter, Ashford, March 26, 1903, æt. 67.

MEDICAL SOCIETY OF THE COUNTY OF CAYUGA.

MEETINGS.—Annual, second Thursday in May; semi-annual, second Thursday in November. Regular meetings the second Thursday in August and February.

Officers. (May, 1903.)

Sheldon Voorhees, *President.* Ledra Heazlit, *Vice-President.*
F. H. Spencer, *Secretary* A. W. Gilmore, *Treasurer.*

Members.

(When no town is mentioned Auburn is to be understood.)

Charles Atwood, Moravia
D. F. Armstrong, 8 Grover st
S. E. Austin, 6½ John st
O. H. Babbitt, 178 Genesee st
M. O. Bentley, Weedsport
F. H. Benedict, Weedsport
C. H. Bennett, 37 Clark st
M. D. Blaine, 16 William st
N. D. Blood, 5 Aurelius av.
E. W. Bogardus, Poplar Ridge
A. H. Brown, 7 William st
B. I. C. Buckland, Fleming
H. D. Chapman, 24 South st
W. S. Cheesman, 22 William st
C. F. Chiles, Montezuma
W. H. Coe, 82 E. Genesee st
W. H. Coe, Jr., 82 E. Genesee st
M. P. Conway, 112 Genesee st
W. T. Cox, Moravia
J. P. Creveling, 22 South st
W. D. Cuddeback, Aurora
Robert Hill Dee, Fair Haven
F. A. Dudley, King Ferry
C. A. Fisher, Victory
A. J. Forman, 55 E. Genesee st
E. G. Fuller, Martville
John Gerin, 68 North st
Amelia Gilmore, 28 South st
J. R. B. Glasgow, 132 Genesee st
Geo. W. Gray, Port Byron
Geo. W. Green, 45 E. Genesee st
A. L. Hall, Fair Haven
Ledra Heazlit, 10 William st
A. F. Hodgman, 26 South st
J. P. Horley, Cato
Frank Hoxie, Union Springs
J. M. Jenkins, 162 Genesee st
Nettie E. Jenkins, 162 Genesee st
A. C. Knapp, Locke
Wm. R. Laird, 97 North st
Charles L. Lang, Meridian
Guy R. Montgomery, 137 South st
B. D. Nellis, Weedsport
F. E. O'Brien, 114 Genesee st
L. F. O'Neil, 6½ William st
B. E. Osborne, 38 Fulton st
Susan G. Otis, 65 Genesee st
F. H. Parker, 156 Genesee st

Frank Putnam, 93 Wall st
R. W. Robinson, 30 Court st
Wm. J. Russell, Aurora
Frank Ryan, Moravia
Frederick Sefton, 137 South st
George Slocum, Genoa
F. H. Spencer, 6 William st
F. C. Smith, Fleming
C. M. Stever, Sennett
A. D. Stewart, Port Byron
A. W. Strohmenger, Kelloggsville
J. D. Tripp, 163 Genesee st
M. B. Van Buskirk, Aurora
Sheldon Voorhees, 88 North st
H. O. Wheeler, King Ferry
J. W. Whitbeck, Cayuga
D. A. White, Montezuma
E. G. Woodruff, 29 William st
Number of Members, 62.

Deceased: B. K. Hoxsie, Jr., Auburn, July 12, 1902, æt. 72.

MEDICAL SOCIETY OF THE COUNTY OF CHAUTAUQUA.

(Organized, 1824; reorganized, 1853.)

MEETINGS.—Annual, second Tuesday in July; semi-annual, second Tuesday in December.

Officers. (July, 1902.)

George E. Blackham, *President.* N. E. Beardsley, *Vice-President.*
Charles Arthur Ellis, *Secretary and Treasurer.*

Censors: Geo. E. Blackham, T. D. Strong, Wm. M. Bemus.

Members.

N. E. Beardsley, Dunkirk
A. J. Bennett, Busti
George E. Blackham, Dunkirk
A. H. Bowers, Jamestown
V. D. Bozovsky, Dunkirk
J. M. Brooks, Jamestown
A. E. Dean, Brocton
A. Wilson Dods, Fredonia
J. J. Drake, Stockton
Charles A. Ellis, Sherman
G. E. Ellis, Dunkirk
R. M. Evarts, Silver Creek
W. J. French, Hamlet
L. C. Green, Panama
V. M. Griswold, Fredonia
Laban Hazeltine, Jamestown
Edw. R. Hopkins, Silver Creek
Frederick C. Hunt, Jamestown
E. A. Jones, Jamestown
J. J. Lenhart, Bemus Point
E. C. Lyman, Jamestown
L. P. McCray, Clymer
J. J. Mahoney, Jamestown
G. D. Marsh, Sherman
MacDonald Moore, Fredonia
W. T. Nash, Irving
Jason Parker, Jamestown
W. A. Putnam, Smith's Mills
C. H. Richards, Dunkirk
N. G. Richmond, Fredonia
R. T. Rolph, Dunkirk
C. A. Rood, Brocton
E. Rood, Westfield
E. A. Schofield, Bemus Point
J. J. Sharp, Silver Creek
O. C. Shaw, Cassadaga
G. E. Smith, Cherry Creek
J. R. Smith, Conewango Valley
W. O. Smith, Falconer
T. E. Soules, Westfield
L. H. Snow, Jamestown
Ellis W. Storms, Cherry Creek
Walter Stuart, Westfield
B. S. Sweetland, Brocton
Walter H. Vosburgh, Dunkirk
W. D. Wellman, Jamestown
J. H. Wiggins, Jamestown
T. C. Wilson, Point Chautauqua
Number of Members, 48.

Deceased: M. N. Benjamin, Dunkirk, November 26, 1902, æt. 59; H. J. Dean, Brocton, October 11, 1901; H. R. Rogers, Dunkirk, October 19, 1901, æt. 79.

MEDICAL SOCIETY OF THE COUNTY OF CHEMUNG.

(Organized May 6, 1836.)

MEETINGS.—Annual, third Tuesday in May; quarterly, third Tuesday in August, November, and February.

Officers. (1903.)

A. H. Baker, *President.* O. J. Bowman, *Vice-President.*
Jessie Louise Herrick, *Secretary.* G. V. R. Merrill, *Treasurer.*

Censors: N. H. Soble, John C. Fisher, Arthur W. Booth, C. W. M. Brown, C. G. R. Jennings.

Members.

(When no town is mentioned Elmira is to be understood.)

F. C. Annabel, 127 E. Hudson st
A. H. Baker, 313 Baldwin st
Arthur W. Booth, 105 N. Main st
O. J. Bowman, Horseheads
C. W. M. Brown, 311 Baldwin st
R. P. Bush, Horseheads
Walter C. Byrne, 257 Baldwin st
C. S. Carey, 212 Lake st
Geo. M. Case, 122 N. Main st
R. R. Chilson, 610 W. Church st
La Rue Colegrove, Wellsburg
W. E. Colegrove, Horseheads
T. A. Dundas, 358 Main st
John C. Fisher, Gleason Sanitarium
W. H. Fisher, 408 S. Main st
Frank H. Flood, 124 Main st
Henry Flood, 403 Lake st
H. H. Ford, Main and 2d st
H. W. Fudge, 375 S. Main st
J. W. Gee, Erin
F. B. Green, 921 Lake st
C. N. Hammond, Elmira Heights
Jessie L. Herrick, 408 Union place
Elizabeth M. Hooper, 467 W. 2d st
Jonas Jacobs, 251 Baldwin st
O. A. Jakway, Breesport
C. G. R. Jennings, 304 E. Church st
Emma C. Le Fevre, 301 W. Clinton st
G. V. R. Merrill, 458 Spaulding st
N. S. Messenger, 558 E. Church st
John Moroney, 328 Baldwin st
J. C. O'Brien, 504 Penn. av
E. A. Reilly, 518 Lake st
Frank W. Ross, 104 N. Main st
Delphine Smith-Butcher, 454 East Church st
G. D. Smith, Wellsburg
N. H. Soble, 458 E. Church st
C. L. Squire, 406 E. Church st
Anna M. Stuart, 418 William st
Sherman Voorhees, 408 Main st
E. H. Wakelee, Big Flats
T. A. Wales, 403 William st
A. J. Westlake, 400 Lake st
J. A. Westlake, 400 Lake st
H. D. Wey, 359 Main st
F. E. Woodhouse, 315 Baldwin st

Number of Members, 46.

MEDICAL SOCIETY OF THE COUNTY OF CHENANGO.

(Organized in 1806.)

MEETINGS.—Annual, second Tuesday in January; semi-annual, second Tuesday in June.

Officers. (January, 1903.)

R. H. Phelps, *President.* H. E. Smith, *Vice-President.*
F. O. Spooner, *Secretary.* S. M. Hand, *Treasurer.*

Censors: Drs. Packer, Gleason, and Hand.

Members.

L. C. Andrews, Pilcher
Louis P. Blair, McDonough
Vincent Burgess, Brisbin
De Witt C. Crumb, South Otselic
L. E. Dixon, S. New Berlin
W. L. Dodge, Afton
George Douglas, Oxford
James B. Drake, Norwich
H. T. Elliot, Greene
R. D. L. Evans, Bainbridge
T. B. Fernald, Norwich
H. S. Gardiner, Hamilton
E. L. Gaus, Oxford
D. A. Gleason, Oxford
O. A. Gorton, Sherburne
J. D. Guy, Chenango Forks
S. M. Hand, Norwich
Blinn A. Harris, Norwich
P. A. Hayes, Afton
J. V. Jacobs, Bainbridge
Reuben Jeffrey, Norwich
George D. Johnson, Oxford
L. M. Johnson, Greene
J. V. Lewis, North Norwich
H. C. Lyman, Sherburne
T. F. Manley, Norwich
E. N. Morgan, S. New Berlin
J. B. Noyes, New Berlin
B. J. Ormsby, Norwich
Thurston G. Packer, Smyrna
Charles B. Palmatier, Plymouth
L. B. Palmiter, Mt. Upton
A. T. Perkins, South Otselic
R. H. Phelps, Norwich
H. E. Smith, Norwich
M. E. Smith, South Edmeston
M. E. Smith, Greene
M. Spencer, Guilford

F. O. Spooner, Sherburne
O. L. Taylor, Bainbridge
R. A. Thompson, Norwich
J. W. Thorp, Oxford
L. A. Van Wagner, Sherburne
L. C. Van Wagner, New Berlin
G. S. Weaver, McDonough
H. H. White, Earlville, Madison Co.
Earl W. Wilcox, Norwich
G. O. Williams, Greene
Number of Members, 48.

MEDICAL SOCIETY OF THE COUNTY OF CLINTON.

(Organized October 6, 1807.)

MEETINGS.—Annual, second Tuesday in January; semi-annual, second Tuesday in July.

Officers. (January, 1903.)

R. B. Lamb, *President.* S. H. Newton, *Vice-President.*
D. S. Kellogg, *Secretary.* F. Madden, *Treasurer.*

Members.

C. W. Arthur, Plattsburgh
Ethan A. Barnes, Plattsburgh
M. D. Briggs, Champlain
W. S. Buck, Plattsburgh
H. E. Centre, Schuyler Falls
W. E. Clough, West Chazy
De Witt C. Dare, Cadyville
L. C. Dodge, Rouse's Point
A. W. Fairbank, Chazy
J. M. Hackett, Champlain
Charles S. Haynes, Redford
O. A. Holcomb, Plattsburgh
W. S. Honsinger, West Chazy
E. S. Howe, Ausable Forks
R. E. Hyde, Plattsburgh
D. S. Kellogg, Plattsburgh
Robert B. Lamb, Dannemora
Elmer E. Larkin, Plattsburgh
A. M. Larkin, Standish
G. E. Letourneau, Rouse's Point
J. H. LaRocque, Plattsburgh
E. M. Lyon, Plattsburgh
J. G. McKinney, Plattsburgh
Frank Madden, Plattsburgh
S. Mitchell, Jr., Saranac
Sanford H. Newton, Rouse's Point
J. B. Ransom, Dannemora
H. H. Reynolds, Malone
John J. Robinson, Saranac
T. Avery Rogers, Plattsburgh
Clarkson C. Schuyler, Plattsburgh
Cassius D. Silver, Plattsburgh
W. U. Taylor, Mooers
Walter Nelson Thayer, Jr., Peru
W. C. Thompson, Plattsburgh
C. B. Vaughan, Morrisonville
D. B. Woodward, Ellenburgh
Number of Members, 37.

Deceased: Samuel Haynes, Saranac, June 27, 1901; E. M. Morehouse, Peru, April 13, 1902.

MEDICAL SOCIETY OF THE COUNTY OF COLUMBIA.

MEETINGS.—Annual, second Tuesday in May, at City Hall, Hudson; semi-annual, first Tuesday in October at Chatham.

Officers. (May, 1903.)

C. G. Rossman, *President.* Z. F. Dunning, *Vice-President.*
T. Floyd Woodworth, *Secretary and Treasurer.*
Censors: E. E. Martin, G. P. K. Pomeroy, N. D. Garnsey, Emmett Niver, H. W. Johnson.

Members.

J. H. Allen, Churchtown
Milford L. Bates, Canaan 4 Corners
Robert W. Bell, Jr., Claverack
O. Howard Bradley, Hudson
T. J. Clelland, Lebanon
J. E. Cochran, Valatie
J. R. Conklin, Philmont
H. W. Cordes, Copake
Henry Cornell, Hillsdale
Z. F. Dunning, Philmont
Crawford E. Fritts, Hudson
John J. Glover, Stuyvesant Falls
N. D. Garnsey, Kinderhook
G. W. E. Goodell, East Chatham
H. W. Johnson, Hudson
J. W. King, Stottville
Martin M. Kittell, Kinderhook
F. T. Kunker, North Chatham

E. E. Martin, Stottville
Nelson E. Mesick, Glenco Mills
R. H. Morey, Old Chatham
Emmett Niver, Hillsdale
Elwood Oliver, Ancram
Wm. P. Platner, Germantown
George P. K. Pomeroy, Stuyvesant
J. F. Rooney, Albany
C. G. Rossman, Hudson
George W. Rossman, Ancram
Charles Skinner, Copake
H. Lyle Smith, Hudson
Wm. O. Smith, Germantown
W. S. Starks, Chatham
Wm. D. Swain, Copake
G. E. Swift, Hudson
G. W. Timmers, Livingston
Sherman Van Ness, Chatham Centre
George W. Vedder, Philmont
Joseph E. Vigeant, Elizaville
I. C. Washburne, Chatham
H. G. Westlake, Hillsdale
John T. Wheeler, Chatham
Thomas Wilson, Hudson
T. F. Woodworth, Kinderhook

Number of Members, 43.

Deceased: A. R. Van Dusen, Claverack, Aug. 21, 1901, æt. 81.

MEDICAL SOCIETY OF THE COUNTY OF CORTLAND.

(Organized August 10, 1808.)

MEETINGS.—Annual, second Thursday in June; semi-annual, second Thursday in December; quarterly, second Thursdays in September and March.

Officers.

Philip M. Neary, *President.* H. T. Dana, *Vice-President.*
Frank D. Reese, *Secretary, Treasurer, and Librarian.*

Censors: H. T. Dana, J. W. Whitney, H. C. Hendrick, F. W. Higgins, F. D. Reese.

Members.

Lucius C. Andrews, Pitcher
I. A. Beach, Cortland
Charles E. Bennett, Cortland
Paul T. Carpenter, Cortland
H. S. Braman, Homer
Henry T. Dana, Cortland
E. A. Didama, Cortland
T. M. Emory, Virgil
Henry Field, Marathon
F. H. Forshee, McGrawville
M. L. Halbert, Cincinnatus
H. C. Hendrick, McGrawville
Francis W. Higgins, Cortland
Frank S. Jennings, Cortland
Benj. Kinyon, Cincinnatus
J. E. Leonard, Hartford Mills
Edward M. McBirney, Willett
Philip M. Neary, Cortland
A. L. Powers, Blodgett Mills
Frank D. Reese, Cortland
M. R. Smith, McGrawville
S. J. Sornberger, Cortland
S. Halsey Stevens, Truxton
H. I. Van Hoesen, Truxton
Charles D. Vernooy, Cortland
Henry Watson, Cincinnatus
J. W. Whitney, Homer

Number of Members, 27.

Deceased: Frank H. Green, Homer, December 7, 1902. æt. 46.

MEDICAL SOCIETY OF THE COUNTY OF DELAWARE.

(Organized July 1, 1806.)

MEETINGS.—Annual, second Tuesday in June; semi-annual, in the Fall.

Officers.

C. S. Allaben, *President.* George L. Hubbell, *Vice-President.*
Stanton Hendrick, *Secretary.* George H. Reynolds, *Treasurer.*

Censors: Thomas Wight, G. C. Smith, H. A. Gates, J. N. Wright.

Members.

Charles S. Allaben, Margaretville
Howard Bell, Delhi
George H. Brinkman, Sidney
Robert Brittain, Downsville
George S. Brown, Margaretville
S. S. Cartwright, Roxbury
S. E. Churchill, Stamford
Thomas L. Craig, Davenport
William J. Cranston, Trout Creek
Gilbert J. Dickson, Bovina

Andrew R. Ellis, Roxbury
E. W. Gallup, Stamford
Henry A. Gates, Delhi
J. A. Gladstone, Andes
Hobart J. Goodrich, Delhi
W. D. Heimer, Hamden
Stanton Hendrick, Treadwell
C. J. Hillis, Griffin's Corners
James A. Holley, Walton
George L. Hubbell, Hobart
Austin D. Johnson, East Branch
E. B. Lake, Merridale
C. G. Lyon, Davenport Centre
E. D. McKenna, Walton
J. S. McNaught, Hobart
W. A. Minor, Bloomville
William B. Morrow, Walton
William Ormiston, Delhi
Lloyd Palmer, East Meredith
Samuel Rabuck, Bovina Centre,
George H. Reynolds, Delhi
John E. Safford, Stamford
Gilbert T. Scott, Davenport
George C. Smith, Walton
Wm. G. Smith, Walton
S. J. White, Franklin
Thomas Wight, Andes
J. N. Wright, Grand Gorge

Number of Members, 38.

MEDICAL SOCIETY OF THE COUNTY OF DUTCHESS.

(Organized in 1806.)

MEETINGS.—Annual, second Wednesday in January, at Poughkeepsie; semi-annual, second Wednesday in June.

Officers. (January, 1903.)

J. E. Sadlier, *President.* H. L. Cookingham, *Vice-President.*
Robert W. Andrews, *Secretary.* D. B. Ward, *Treasurer.*
Censors: Robert K. Tuthill, E. H. Gerow.

Members.

John H. Acheson, Poughkeepsie
W. S. Ackert, Poughkeepsie
H. E. Allison, Matteawan
Robert W. Andrews, Poughkeepsie
B. N. Baker, Rhinebeck
Thomas E. Bamford, State Hospital, Poughkeepse
Edwin Barnes, Pleasant Plains
H. Weston Barnum, Poughkeepsie
T. J. Barton, Red Hook
Guy C. Bayley, Poughkeepsie
Nelson Borst, Poughkeepsie
J. N. Boyce, Stanfordville
E. M. Burns, Poughkeepsie
John A. Card, Poughkeepsie
W. J. Cavanaugh, State Hospital, Poughkeepsie
J. Stuart Chaffee, Asst. Surgeon, U. S. A., Plattsburgh
W. M. Clark, Matteawan
W. J. Conklin, Fishkill
Isaac M. Cornell, Wappinger's Falls
James M. Cronk, Hyde Park
Thos. K. Cruse, Wappinger's Falls
John H. Cotter, Poughkeepsie
John H. Cotter, Pine Plains
W. G. Dobson, Poughkeepsie
J. H. Doughty, Matteawan
Austin T. Fink, Freedom Plains
Elizabeth H. Gerow, Poughkeepsie
H. A. Gibbon, Poughkeepsie
J. R. Grant, Poughkeepsie
Edward L. Hanes, State Hospital, Poughkeepsie
R. A. Hoyt, Fishkill
I. G. Harris, State Hospital, Poughkeepsie
George Huntington, La Grangeville
C. W. Ivie, La Grangeville
M. Julien, Pleasant Valley
Grace N. Kimball, Poughkeepsie
T. B. Kinkead, Poughkeepsie
D. A. Knapp, North Clove
Charles H. Langdon, Poughkeepsie
I. D. Leroy, Pleasant Valley
Mark M. Lown, Rhinebeck
D. H. MacKenzie, Mabbettsville
Frederick J. Mann, Poughkeepsie
Lewis H. Marks, Poughkeepsie
C. H. McCambridge, Poughkeepsie
S. F. Mellen, State Hospital, Poughkeepsie
Raymond T. Metcalf, Poughkeepsie
J. E. Moith, Fishkill
Franklyn Murphy, Staatsburg
C. W. Pilgrim, State Hospital, Poughkeepsie
J. O. Pingry, Mill Brook
J. W. Poucher, Poughkeepsie
Horace R. Powell, Poughkeepsie
Churchill A. Pritchard, Tivoli
Emma Putnam, State Hospital, Poughkeepsie
Selwyn A. Russell, Poughkeepsie

J. E. Sadlier, Poughkeepsie
S. M. W. Scott, Matteawan
D. W. Sheedy, Poughkeepsie
D. C. Tripp, Beekmanville
C. H. Tripp, Clinton Corners
R. K. Tuthill, Poughkeepsie
G. A. Van Wagner, Wappinger's Falls
D. B. Van Wyck, Manchester Bridge
Louis T. Waldo, State Hospital, Poughkeepsie
D. B. Ward, Poughkeepsie
W. S. Watson, Matteawan
F. B. Weaver, Hyde Park
Henry D. White, Hopewell Junction
Howell White, Fishkill
H. C. Wilbur, Pine Plains
George H. Williams, Fishkill
J. S. Wilson, Poughkeepsie
L. C. Wood, Poughkeepsie

Number of Members, 74.

Deceased: M. T. Pultz, Stanfordville, September 9, 1902, æt. 59; G. M. Wellman, Dover Plains, January 13, 1902, æt. 65; John G. Elliott, Poughkeepsie, May 12, 1902; Alfred Hasbrouck, Poughkeepsie, May 9, 1903, æt. 83.

MEDICAL SOCIETY OF THE COUNTY OF ERIE.

MEETINGS.—Annual, second Tuesday in January; semi-annual, second Tuesday in June.

Officers. (1903.)

Ernest Wende, *President.* John D. McPherson, *Vice-President.*
Franklin C. Gram, *Secretary.* Edward Clark, *Treasurer.*
Wm. C. Callanan, *Librarian.*

Censors: J. B. Coakley, Henry Lapp, F. E. Fronczak, H. R. Hopkins, Irving W. Potter.

Members.

(Where no town is mentioned Buffalo is to be understood.)

George Abbott, Hamburg
Arthur B. Allen, 413 E. Utica
Thomas G. Allen, 419 Elk st
Chas. H. W. Auel, 277 High st
Stanley C. Babcock, 2319 Main st
John E. Bacon, 79 Niagara sq
Lucius L. Ball, 190 Dearborn st
Edward H. Ballou, Gardenville
Rollin L. Banta, 358 S. Division st
Edwin R. Barnes, 1258 West av
William C. Barrett, 208 Franklin st
T. L. Barry, Willink
Bernard Bartow, 481 Delaware av
Theodore V. Bauer, 735 Washington
A. W. Bayliss, 592 Spring st
A. L. Benedict, 174 Franklin st
A. G. Bennett, 90 W. Chippewa st
Henry G. Bentz, 894 Michigan st
W. D. Bidaman, 335 Connecticut st
Henry H. Bingham, 2485 Main st
Elias S. Bissell, 2793 Main st
William G. Bissell, 143 Elmwood av
Loren F. Bois, 286 Norwood av
John D. Bonner, 144 Jewett av
C. R. Borzilleri, 22 Morgan
Bentley S. Bourne, Hamburg
Charles W. Bourne, Hamburg
Carlos F. Bowman, Alden
Clara E. Bowen, 1223 Lovejoy st
F. E. L. Brecht, 361 William st
Albert H. Briggs, 267 Hudson av
Elmer E. Briggs, 879 Clinton st
Mark N. Brooks, Springville
Ira C. Brown, 411 14th st
H. B. Brownell, 1430 Main st
Frank A. Burghardt, 632 Elm st
Joseph Burke, 208 N. Division st
L. Burrows, 388 Franklin st
Henry C. Buswell, 868 Main st
Wm. C. Callanan, 190 Front av
T. B. Carpenter, 142 N. Pearl
Evangeline Carroll, 285 Ashland av
Jane W. Carroll, 285 Ashland av
Henry T. Carter, 210 Hoyt st
Charles Cary, 340 Delaware av
Wm. H. Chace, 266 Connecticut st
John S. Champlain, 1872 Niagara st
N. V. Chappell, 418 Breckenridge st
J. W. Charters, 569 Walden av
Edward Clark, 845 Ellicott sq
Mary Clayton, 44 Elmwood av
Chas. A. Clements, 312 Sycamore st
J. C. Clemesha, 329 Franklin st
Wm. P. Clothier, 1126 Main st
John B. Coakley, 339 Delaware av
Bernard Cohn, 540 Niagara st
A. E. Collins, 248 Dearborn st
A. J. Colton, 27 E. Ferry st
Chas. E. Congdon, 1034 Jefferson st
D. J. Constantine, 528 West av
Edward J. Cook, 1620 Broadway
George F. Cott, 531 Mooney Bldg
Floyd S. Crego, 469 Delaware av
Montgomery A. Crockett, 452 Franklin st
C. Julia Cummings, 560 Auburn av

Byron H. Daggett, Ellicott sq
John Dambach, 417 Michigan st
John H. Daniels, 184 Genesee st
W. J. Deane, 134 E. Ferry st
L. A. Denton, 445 Franklin st
Mary J. Denton, 445 Franklin st
Alfred E. Diehl, 361 Pearl st
Conrad Diehl, 32 W. Genesee st
E. M. Dooley, 406 Louisiana st
L. Bradley Dorr, 300 Jefferson st
J. Henry Dowd, 378 Franklin st
Wm. Dowlmen, 536 Swan st
Daniel P. Doyle, 217 Franklin st
S. A. Dunham, 72 W. Chippewa st
Thomas F. Dwyer, 89 West av
Fred'k H. Ehinger, Ebenezer
David C. Eisbein, 399 Broadway
Chalers P. Eller, 1440 Jefferson st
Henry S. Ellwood, 9 W. Mohawk st
Alfred F. Erb, Clarence, N. Y.
Clark E. Ernest, 282 W. Ferry st
J. Glen Ernest, 386 Walden av
E. N. Ewell, Lancaster, N. Y.
George E. Fell, 72 Niagara st
F. H. Field, Depew
John J. Finnerty, 429 Franklin st
John D. Flagg, 384 Franklin st
Edgar A. Forsyth, 326 Franklin st
Joseph Fowler, 141 Delaware av
C. C. Frederick, 64 Richmond av
Jane N. Frear, 28 Orton place
F. E. Fronczak, 508 Fillmore av
Edward L. Frost, 489 Plymouth av
Maud J. Frye, 224 Allen st
Wm. H. Gail, East Aurora
E. J. Gilray, Erie County Hospital
Frank M. Gipple, Bowmansville
S. Goldberg, 584 N. Division st
Franklin C. Gram, 460 Glenwood av
John H. Grant, 176 Virginia
H. J. Grant, 302 Bouck av
John R. Gray, 224 Front av
Stephen S. Greene, 328 Niagara st
De Witt C. Greene, 1125 Main st
Walter D. Greene, 385 Jersey av
Willis G. Gregory, 530 Main st
J. W. Grosvenor, 118 Plymouth av
Benjamin H. Grove, 334 Pearl st
R. K. Grove, 334 Pearl st
Adelbert G. Guamaer, 802 Bailey av
Robert S. Hambleton, Kenmore
Lawrence G. Hanley, 696 Seneca st
Robert Hebenstreit, 430 Broadway
Devillo W. Harrington, 1430 Main st
John T. Harris, Tonawanda
Marcell Hartwig, 884 Main st
John Hauenstein, 309 Elmwood av
Herman E. Hayd, 493 Delaware av
Frederick W. Hayes, 91 Broadway
Geo. J. Hearne, 191 Auburn av
F. E. Hill, 532 Swan st
G. J. Himmelsbach, 137 W. Tupper st
Felix Hintz, 1037 Ellicott
F. Whitehill Hinkel, 305 Delaware av
John A. Hoffmeyer, 150 East st
Henry R. Hopkins, 433 Franklin st
Patrick H. Hourigan, 739 Seneca st
Charles F. Howard, 1415 Main st
Lucien Howe, 183 Delaware av
Stephen Y. Howell, 164 Franklin st
Burt P. Hoyer, 216 N. Division st
F. F. Hoyer, Tonawanda
Alvin A. Hubbell, 212 Franklin st
Howard L. Hunt, Orchard Park
Mary M. Huntley, 369 Hudson st
Arthur W. Hurd, State Hospital
Henry D. Ingraham, 405 Franklin st
Geo. N. Jack, Depew
Wm. H. Jackson, Springville
C. R. Jennings, Alden
Wm. E. Jennings, Boston
Carlton R. Jewett, 1299 Main st
Charles S. Jewett, 892 Main st
B. C. Johnson, 1404 Main st
Thomas M. Johnson, 161 E. Ferry st
Ray H. Johnson, 180 N. Division st
Wm. C. Jolls, Orchard Park
Allen A. Jones, 436 Franklin st
Andrew Kamerling, 171 Bryant st
Catharine E. Kelley, 45 Cleveland av
Hiram A. Kendall, 786 Elmwood av
John Ketchum, 302 Hampshire st
E. J. Kiepe, 618 Main st
A. B. Knisley, 356 Swan st
Edward E. Koehler, 733 Broadway
Jacob M. Krauss, 891 Michigan st
Wm. C. Krauss, 371 Delaware av
Lewis Krombein, 246 Elm st
Julius F. Krug, 870 Broadway
Helena Kuhlman, State Hospital
Henry Lapp, Clarence
Henry R. Lapp, Clarence
Cora B. Lattin, 156 Humboldt
Henry W. Lattin, 156 Humboldt
Prescott Le Breton, 122 N. Pearl st
H. C. Leonhardt, Tonawanda
Benjamin G. Long, 520 Elmwood av
Eli H. Long, 1335 Main st
C. E. Long, 192 Richmond av
Henry E. Long, 393 Hampshire st
R. H. Lonsbury, 735 Elmwood av
Benj. L. Lothrop, 81 Breckenridge st
Earl P. Lothrop, 153 Delaware av
Emil Lustig, 604 William st
Samuel H. Lynde, 318 Auburn av
U. C. Lynde, 241 Swan st
A. T. Lytle, 260 Lexington av
J. W. Maguire, 1633 Jefferson st
Matthew D. Mann, 37 Allen st
Marion Marsh, 152 Woodlawn
Andrew J. Martin, Clarence
H. G. A. Matzinger, 519 Franklin st

Wm. B. May, 658 Clinton st
Albert H. McBeth, 35 W. Eagle st
J. J. McCullough, Ellicott sq
Wm. A. McFarlane, Springville
Geo. H. McMichael, 75 W. Tupper
George W. McPherson, Lancaster
John D. McPherson, Akron
H. Mead, 758 Elmwood av
John G. Meidenbauer, 291 Maple st
F. T. Metcalf, 329 Franklin st
William Meisburger, 355 Genesee
Reuben S. Myers, Clarence Centre
E. J. Meyer, 1312 Main st
Herbert Mickle, 526 Delaware av
John Middleton, 233 Massachusetts st
Jacob Miller, 1115 Genesee st
John G. Miller, Lancaster
F. H. Milliner, 5 N. Pearl
Jas. J. Mooney, 393 Seventh st
David A. Morrison, 509 Virginia st
Henry J. Mulford, 466 Franklin st
Katherine Munhall, 378 Auburn av
Henry B. Murray, Tonawanda
Jas. W. Nash, 422 Elk
E. C. W. O'Brien, 1195 Main
Francis M. O'Gorman, 1298 Jefferson
A. T. O'Hara, 770 E. Ferry st
Wm. J. Packwood, 1280 Michigan av
Roswell Park, 510 Delaware av
L. P. L. Parker, Akron
John Parmenter, 372 Franklin st
R. L. Patterson, 537 Franklin st
George W. Pattison, 22 Chippewa st
John A. Pettit, 519 Swan st
Edward N. Pfohl, 539 Main st
Wm. C. Phelps, 146 Allen st
H. Arnold Pierce, Blasdell
Gustav A. Pohl, 96 Lemon st
Julius Pohlman, 382 Franklin st
Luke E. Pomeroy, 187 Masten st
James S. Porter, 289 Cedar st
Irving W. Potter, 806 Fillmore av
Julius H. Potter, 177 Dearborn st
Samuel Potter Lancaster
Wm. W. Potter, 284 Franklin st
Fred. H. Powell, 179 Franklin st
Frederick Preiss, 160 Franklin st
William Preiss, 160 Franklin st
John H. Pryor, 56 Allen st
James W. Putnam, 525 Delaware av
Lillian C. Randall, 217 Franklin st
H. M. Reinhardt, 857 Jefferson st
W. Scott Renner, 361 Pearl st
Charles J. Reynolds, 322 Fox st
Francis M. Rich, 284 Richmond av
Charles A. Ring, 364 Niagara st
William G. Ring, 364 Niagara st
DeLancey Rochester, 469 Franklin st
Benjamin F. Rogers, 221 Franklin st
Reuben M. Root, 537 William st
R. R. Ross, Buffalo General Hospital
E. J. Rulison, 37 Elmwood av
Clinton A. Sage, 224 W. Ferry st
Albert Satterlee, 868 Niagara st
C. A. Schladermundt, 510 William st
Ludwig Schroeter, 529 Fillmore av
Macy B. Searls, East Aurora
D. E. Shelden, 433 Breckenridge st
Hewitt H. Sherman, 666 Main st
C. S. Siegfried, 378 Franklin st
Duncan Sinclair, Tonawanda
Wm. H. Slacer, 49 Niagara st
C. M. Smith, 494 Massachusetts st
Chauncey P. Smith, 90 N. Pearl st
Eugene A. Smith, 1018 Main
Edward T. Smith, 189 14th st
James S. Smith, 66 High st
Louis G. Smith, 300 Elk st
Irving M. Snow, 476 Franklin st
Frederick H. Stanbro, Springville
Loren H. Staples, 121 E. Ferry st
Elmer Starr, 523 Delaware av
Charles G. Stockton, 436 Franklin st
James Stoddard, 162 14th st
Brayton N. Strong, Boston
O. C. Strong, Coldon
Ransford C. Tabor, Tonawanda
Fridolin Thoma, 1072 Lovejoy st
John C. Thompson, 55 Amherst st
Justin G. Thompson, Angola
Wm. H. Thornton, 570 Niagara st
Emil S. Tobie, 41 E. Mohawk st
H. S. Townsend, 1177 Seneca st
G. R. Trowbridge, 1331 Main st
Hiram P. Trull, Williamsville
E. H. Tweedy, 394 Vermont st
John J. Twohey, 301 Masten st
C. A. Tyler, Alden
Julius Ullman, 400 Franklin st
Adolph H. Urban, 523 Franklin st
P. W. Van Peyma, 445 William st
W. Van Peyma, 1294 Fillmore st
Charles A. Wall, 306 Hudson st
John J. Walsh, 544 Ellicott st
Walden M. Ward, North Collins
F. L. Watkins, 13 Williams st
A. L. Weil, 291 Maple st
Chris. A. Weinbach, 1773 Genesee st
Ernest Wende, 471 Delaware av
Geo. W. Wende, 471 Delaware av
Julius Wenz, Lancaster
Geo. H. Westinghouse, 541 Eagle
Mary B. Wetmore, 30 Woodlawn av
Samuel W. Wetmore, 30 Woodlawn av
Charles H. Wetzel, 697 Jefferson st
Electa B. Whipple, 491 Porter av
John E. Whitmore, 1099 Genesee st
J. F. Whitwell, 368 Swan st
Herbert U. Williams, 221 North st
Edw. R. Wise, Williamsville
Albert B. Wilson, 51 Eastwood pl
James P. Wilson, 867 Niagara st

N. W. Wilson, 153 7th st
A. E. Woehnert, 436 Franklin st
Wm. H. Woodbury, 991 Williams st
John W. Woodruff, 17 Unger av
Cornelius C. Wyckoff, 482 Delaware av
George W. York, 190 Franklin st
Edson H. Young, 444 Elk st
Number of Members, 317.

Deceased: Clayton M. Daniels, Buffalo, Sept. 6, 1902, æt. 48; Wm. C. Fritz, Buffalo, Mar. 31, 1903, æt. 36; Thomas Lothrop, Buffalo, Aug. 7, 1902, æt. 66; Herman Mynter, Buffalo, Feb. 9, 1903, æt. 57; Eugene E. Storck, Buffalo, Sept. 10, 1902, æt. 47.

MEDICAL SOCIETY OF THE COUNTY OF ESSEX.

ANNUAL MEETING.—Third or fourth Tuesday in May.
(Society suspended.)

MEDICAL SOCIETY OF THE COUNTY OF FRANKLIN.

(Organized in 1814; reorganized in 1848.)

MEETINGS.—Annual, first Tuesday in January; semi-annual, first Tuesday in June; quarterly, first Tuesday in September.

Officers. (January, 1903.)

E. S. McClellan, *President.* S. D. Williamson, *Vice-President.*
R. J. Wilding, *Secretary and Treasurer.*
Censors: E. M. Austin, G. H. Oliver, W. N. MacArtney.

Members.

W. A. Atwater, S. Regis Falls
E. M. Austin, Tupper Lake
E. R. Baldwin, Saranac Lake
J. O. A. Beaupre, Malone
J. W. Blackett, Fort Covington
L. C. Brunet, Brushton
C. Crippen, Trout River
P. F. Dolphin, Malone
H. Funness, Malone
J. A. Grant, Malone
W. H. Harwood, Chasm Falls
J. D. Harrigan, Chateaugay
G. Howe, Chateaugay
C. A. Hastings, Constable
J. A. Johnson, Chateaugay
H. N. Kinghouse, Saranac Lake
E. A. La Rocque, Malone
W. N. MacArtney, Fort Covington
F. Markle, Bangor
E. S. McClellan, Saranac Lake
C. McConnell, Hogansborough
A. E. Moody, Dickinson Centre
J. W. Morarity, Cherubusco
R. L. Morgan, Tupper Lake
W. S. Nelson, Saranac Lake
G. H. Oliver, Malone
C. E. Pearl, North Bangor
H. H. Reynolds, Malone
H. S. Rockwood, Bombay
E. A. Rust, Moira
G. M. Sabin, Malone
C. Skinner, Malone
J. A. Smart, Fort Covington
C. B. Smith, West Bangor
E. E. Thurber, Brainardville
E. L. Trudeau, Saranac Lake
C. C. Trembly, Saranac Lake
J. S. Van Vechten, Chateaugay
W. M. Warren, Lake Placid
F. D. Whitehead, Burke
W. A. Wardner, St. Regis Falls
C. F. Wicker, Saranac Lake
A. G. Wilding, Malone
R. J. Wilding, Malone
S. D. Williamson, Malone
Number of Members, 45.

MEDICAL SOCIETY OF THE COUNTY OF FULTON.

MEETINGS.—Annual, second Thursday in January; semi-annual, second Thursday in June.

Officers.

J. J. Beard, *President.* E. C. Podvin, *Vice-President.*
M. Somers, *Secretary.* M. F. Drury, *Treasurer.*
Censors: Wm. Davis, C. M. Lefler, C. B. Walrad.

Members.

Eugene Beach, Gloversville
J. J. Beard, Gloversville
Frank Beebe, Johnstown
S. L. Clemens, Gloversville
L. J. Daily, Gloversville
William Davis, Gloversville
M. F. Drury, Gloversville
John Edwards, Gloversville
L. R. Ellithorpe, Johnstown
Nelson Everest, Gloversville
H. C. Finch, Broadalbin
W. S. Garnsey, Gloversville
E. H. Goodfellow, Gloversville
J. A. Hagar, Gloversville
A. C. Hagedorn, Gloversville
Mary Hopkins, Gloversville
R. H. Howland, Broadalbin
A. L. Johnson, Gloversville
J. W. Joslin, Johnstown
C. M. Lefler, Gloversville
George Lenz, Gloversville
W. C. McCulloch, Gloversville
Charles B. Mosher, Johnstown
Chas. Pannaci, Gloversville
E. C. Podvin, Johnstown
Robert Palmer, Gloversville
F. W. Shaffer, Gloversville
M. Somers, Johnstown
David B. Still, Johnstown
J. K. Thorne, Gloversville
J. D. Vedder, Johnstown
C. B. Walrad, Johnstown
W. C. Wood, Gloversville
James K. Young, Johnstown

Number of Members, 34.

MEDICAL SOCIETY OF THE COUNTY OF GENESEE.

(Organized about 1811.)

MEETINGS.—Annual, second Tuesday in June; semi-annual, second Tuesday in January, at Batavia.

(Society at present not in active existence.)

MEDICAL SOCIETY OF THE COUNTY OF GREENE.

(Organized July, 1806: Incorporated June 14, 1896.)

MEETINGS.—Annual, second Tuesday in May; semi-annual, second Tuesday in October; quarterly, second Tuesday in July and January.

Officers. (1903.)

Geo. Haner, *President.*
Robert Selden, *Secretary.*
P. G. Waller, *Vice-President.*
C. E. Willard, *Treasurer.*

Members.

Ambrose Beach, Coxsackie
Rudolph Bestle, Hunter
Charles H. Chubb, Palenville
George Conkling, Durham
F. S. Deyo, Hunter
George A. Deyo, Westkill
E. E. Elliott, Catskill
Luther Emerick, Catskill
Nelson Fanning, Catskill
Edwin L. Ford, Lexington
S. L. Ford, Hensonville
Andrew H. Getty Athens
Nelson H. Griffin, Cairo
George Haner, Tannersville
S. A. Holcomb, Palenville
L. B. Honeyford, Cairo
W. F. Lamont, Catskill
Bradley S. McCabe, Greenville
Charles P. McCabe, Greenville
H. M. Mace, Catskill
J. H. Mead, Hunter
William H. Mead, Windham
E. E. Maryott, Coxsackie
E. H. Merriman, Coxsackie
Chas. D. Mulberry, Windham
Daniel D. Parrish, New Baltimore
J. B. Rouse, Leeds
L. Safford, East Durham
Robert Selden, Catskill
I. T. Sutton, Prattsville
C. H. Turner, Ashland
Edmund C. Van Dusen, Athens
A. W. Van Slyke, Coxsackie
Percy G. Waller, New Baltimore
W. A. Wasson, Greenville
Charles E. Willard, Catskill

Number of Members, 35.

Deceased: Isaac J. Van Hoesen, Medway, August 7, 1901, æt. 68; Peter J. Stanley, Windham, Oct. 4, 1901, æt. 76.

MEDICAL SOCIETY OF THE COUNTY OF HERKIMER.

(Organized August 5, 1806.)

MEETINGS.—Annual, first Tuesday in March at the Court-house in Herkimer; semi-annual, first Tuesday in September; quarterly, first Tuesday in June and December. The place for all meetings, except the annual, is designated at the annual meeting.

Officers. (March, 1903.)

C. B. Trafford, *President.* George M. McComb, *1st Vice-President.*
U. G. Williams, *2d Vice-President.* A. C. Douglass, *3d Vice-President.*
A. Walter Suiter, *Secretary.* George Graves, *Treasurer.*
William P. Earl *Librarian.*

Committee on Hygiene: A. B. Santry, Clark Getman, Cyrus Kay, I. S. Edsall, W. E. Hayes.

Committee on Microscopy: W. D. Garlock, *Chairman;* O. H. Deck, A. Walter Suiter.

Committee on Program: George M. McCombs, S. S. Richards, W. E. Hayes.

Committee on Legislation: The President, Secretary and Treasurer, *ex officio.*

Members.

George M. Armstrong, West Winfield
F. M. Barney, Dolgeville
M. W. Brown, Cedarville
W. W. Budlong, Frankfort
Maynard G. Burgess, Herkimer
J. E. Casey, Mohawk
F. B. Casey, Mohawk
H. J. Cristman, Columbia
Fred F. Comstock, Ilion
John L. Crofts, Little Falls
O. H. Deck, Herkimer
D. M. Devendorf, Herkimer
Lyman C. Dexter, Newport
A. J. Douglass, Ilion
A. O. Douglass, Little Falls
A. C. Douglass, Ilion
E.gar H. Douglas, Little Falls
E. M. Draper, Ilion
William P. Earl, Little Falls
I. S. Edsall, Middleville
John B. Ellis, Little Falls
George S. Everleth, Little Falls
J. D. Fitch, Mohawk
George Graves, Herkimer
Wm. D. Garlock, Little Falls
Clark Getman, Dolgeville
Charles H. Glidden, Little Falls
H. H. Greene, Paine's Hollow
Frederic J. Harter, Herkimer
Wm. E. Hayes, Frankfort
Ward E. Hunt, Little Falls
Stephen A. Ingham, Little Falls
Cyrus Kay, Herkimer
Wm. G. Lewis, West Schuyler
Miles Longshore, Cold Brook
George M. McCombs, Frankfort
Carl C. Mann, Ilion
Adam Miller, Jordanville
Irving O. Nellis, Herkimer
Stuart W. Nelson, Old Forge
T. B. O'Neil, Ilion
George P. Rasback, Mohawk
James I. Rasback, Ilion
S. S. Richards, Frankfort
A. B. Santry, Little Falls
John H. Shaper, Herkimer
John H. Stephens, West Winfield
C. G. Strobel, Dolgeville
A. Walter Suiter, Herkimer
Edgar C. Swift, Jordanville
William Tibbits, Newville
C. B. Trafford, Middleville
D. P. Van Court, Mohawk
Delavan E. Walker, Ilion
Robert W. Warner, Ilion
U. Grant Williams, Newport
William H. Young, Starkville

Number of Members, 57.

Deceased: J. B. Holcomb, Newport; John D. Young, Starkville.

MEDICAL SOCIETY OF THE COUNTY OF JEFFERSON.

MEETINGS.—Annual, second Tuesday in January; semi-annual, second Tuesday in July; quarterly, second Tuesday in April and October.

Officers. (January, 1903.)

J. A. Barnett, *President.* Richard L. Gray, *Vice-President.*
F. R. Calkins, *Secretary.* C. M. Rexford, *Treasurer.*

Censors: O. C. Eastman, D. C. Rodenhurst, M. A. Vincent, C. F. Adams, J. M. Crawe.

Members.

C. F. Adams, Carthage
Fred. C. Bailey, Adams Centre
Wm. C. Bailey, Adams Centre
J. A. Barnett, Watertown
C. N. Bibbins, Watertown
H. D. Bingle, Carthage
Wallace N. Brown, Watertown
H. M. Buchanan, St. Lawrence
R. J. F. Burton, Depauville
F. R. Calkins, Watertown
Gilbert Cannon, Watertown
C. A. Catlin, Redwood
E. A. Chapman, Belleville
B. C. Cheesman, Watertown
William C. Couch, Ox Bow
J. M. Crawe, Jr., Watertown
Eugene M. Crabb, Cape Vincent
F. T. Dale, Depauville
Siegfroid Dandurand, Watertown
H. H. Deane, Watertown
A. J. Dick, Watertown
C. S. Drury, Natural Bridge
Charles Douglas, Black River
O. C. Eastman, Watertown
E. E. Eddy, Redwood
W. C. Fawdry, Lorraine
G. A. Foote, Dexter
C. B. Forsythe, Alexandria Bay
J. T. Fowkes, Lafargeville
H. J. Frame, Clayton
S. W. Frame, Belleville
Geo. F. Gardiner, Pierrepont Manor
L. E. Gardner, Black River
A. A. Getman, Chaumont
A. W. Goodale, Watertown
Alfred Goss, Adams
J. C. Graham, Philadelphia
Richard L. Gray, Clayton
G. D. Gregor, Watertown
H. A. Hoyt, Watertown
L. N. Hurlbut, Watertown
F. A. Hutchins, Antwerp
G. H. Ives, Watertown
P. H. Johnson, Adams
J. E. Jones, Evans Mills
Albert A. Joslin, Watertown
Wm. J. Kellow, Watertown
James E. Kelsey, Theresa
C. C. Kimball, Watertown
H. L. Ladd, Sacket Harbor
O. J. La Fontaine, Chaumont
M. S. Lord, Sacket Harbor
Sheldon D. Lord, Sacket Harbor
James F. McCaw, Watertown
E. R. McCreary, Watertown
Wm. N. Maloney, Three-Mile-Bay
Lois F. Mansfield, Watertown
Thomas Masson, Cape Vincent
F. W. H. Massey, Brownville
Isabel M. Meader, Watertown
S. L. Merrill, Carthage
John M. Mills, Clayton
A. L. Morgan, Dexter
Charles Parker, Three-Mile-Bay
F. C. Peterson, Watertown
John Pierce, Adams
H. C. Potter, Mannsville
C. M. Rexford, Watertown
DeWitt C. Rodenhurst, Philadelphia
George G. Sabin, Watertown
C. J. Severance, Mannsville
Florence A. Sherman, Watertown
W. H. H. Silas, Ellisburg
F. B. Smith, Watertown
H. H. Smith, Watertown
J. Monroe Smith, Watertown
M. L. Smith, Watertown
H. L. Smith, Rodman
Gordon P. Spencer, Watertown
James V. Spencer, Watertown
A. B. Stevens, Watertown
Harold B. Stowel, Lafargeville
O. O. Stowell, Watertown
J. R. Sturtevant, Theresa
George E. Sylvester, Black River
W. G. Terry, Henderson
A. S. Thompson, Ellisburg
Arthur L. Tinkess, Plessis
W. A. Vincent, Three-Mile-Bay
E. E. Ward, Pamelia Four Corners
Ervin W. Witt, Brownville
G. H. Wood, Antwerp
E. S. Willard, Watertown

Number of Members, 93.

MEDICAL SOCIETY OF THE COUNTY OF KINGS.

MEETINGS.—Annual, third Tuesday in January; stated meeting, third Tuesday of other months; all meetings are held in Brooklyn, at the Society Building, 1313 Bedford avenue. A "Journal and Reading-Room" is open daily (except Sundays) from 10 A. M. to 10 P. M. The Society also owns and publishes monthly *The Brooklyn Medical Journal.*

Officers. (January, 1903.)

Charles N. Cox, *President.*
John E. Sheppard, *Vice-President.*
William S. Hubbard, *Secretary.*
Wm. C. Woolsey, *Associate Secretary.*
O. A. Gordon, *Treasurer.*
John R. Stivers, *Associate Treasurer.*
J. M. Winfield, *Directing Librarian.*

Censors: J. R. Morrison, Walter C. Wood, William F. Dudley, J. M. Van Cott, H. G. Webster.

Trustees: Charles Jewett, George R. Fowler, Joseph H. Hunt, William Browning, Henry A. Fairbairn.

Active Members.

(Where no town is mentioned Brooklyn is to be understood.)

Acetta, Michele, 105 Sands st
Adams, Henry Frederick, 32 Palmetto st
Addoms, Louis P., 272 Halsey st
Ager, Louis Curtis, Stilman Pl. and Third Av., B. R.
Alderton, Henry A., 142 Clinton st
Aldridge, Vincent, 32 Clarkson st
Allan, J. Glen, 141 Noble st
Alleman, L. A. W., 64 Montague st
Amador, Martin, 187 Park av
Ambler, Alfred S., New Jersey
Anderson, Alexander L., 527a Sixth av
Anderson, Louis N., 513 Bedford av
Anderson, Robert Burns, 765 Nostrand av
Andrew, Joseph H., 500 Madison st
Applegate, W. S., 931 Flatbush av
Arbona, Antonio, 128 Pennsylvania av
Arrowsmith, H., 180 Clinton st
Ayres, Benjamin, 213 Jefferson av
Babcock, E. Howard, D.D.S., 85th st and 2nd av
Babington, John J., 40 Somers st
Bacon, Charles Bowman, Cumberland Street Hospital
Baguely, Henry B., 29 Debevoise st
Bailey, Charles A., 863 Jefferson av
Bailey, Frederick D., 260 Hancock st
Baker, Frank Russell, 540 Bedford av
Balaban, Sigbert, 35 Palmetto st
Baldwin, Frank, 691 Willoughby av
Baldwin, Janette, 123a Hart st
Baldwin, L. Grant, 28 Schermerhorn st
Barber, Calvin F., 57 S. Oxford st
Barber, Vincent, 238 Arlington av
Bartlett, Homer L., 88 Fenimore st
Bartley, Elias H., 21 Lafayette av
Bayles, Havens Brewster, 125 Seventh av
Beasley, Crawford D., 702 Greene av
Beatty, George W., 501 Lafayette av
Becker, James P., 473 Madison st
Beebe, Frances Peele, 145 Clinton st
Beers, Nathan T., 1265 Bedford av
Belcher, William N., 25 S. Portland av
Bell, Alfred, 37 Linden st
Bell, Henry Edwin, 428 Park pl
Bell, Robert J., 304 Warren st
Bell, Rosalie, 655 Carroll st
Bellows, Charles M., 442 Nostrand av
Bender, Herman P., 683 Bushwick av
Bennett, Edward C., 249 58th st
Bennett, Franklin, 686 Greene av
Benton, Stuart H., 720 Nostrand av
Berendsohn, W. A., 598 Carlton av
Berlenbach, Phillip H., 9 Stuyvesant av
Bermingham, Francis H., 133 Remsen st
Bernauer, E. A., 157 Tompkins av
Berwirth, Julius C., 253 Henry st
Biggam, William H., 1197 Dean st
Billings, Frank S., 396 Franklin av
Bird, James R., 247 Gates av
Blackmar, Bruce G., Ovington near Third av
Blaisdell, S. C., 500 Bedford av
Blake, James A., 352 Jefferson av
Bodkin, Martin L., 290 Clinton av
Boes, William, 200 Graham av
Bogart, Arthur H., 139 Seventh av
Bogart, J. Bion, 423 Washington av
Boggs, Seth D., 407 Jefferson av
Bowron, Francis W., 259 Tompkins av
Boyden, Frank Edward, 622 Marcy av
Braislin, William C., 217 St. James pl
Brewster, Richard C., 126 Lefferts pl
Briggs, Benjamin M., 106 Willoughby st
Brinsmade, William B., 117 Montague st
Bristow, Algernon Thomas, 234 Clinton st
Brown, Annie M., 155 Halsey st
Brown, Lucy Hall, 158 Montague st
Brown, Samuel S., 844 Lafayette av
Brown, Thomas E., 278 Clinton av
Browning, William, 54 Lefferts pl
Brundage, Albert H., 1073 Bushwick av
Brush, Arthur C., 29 S. Portland av
Brush George W., 462 Ocean av
Bryan, Walter, 173 Amity st
Bryn, Harold, 384 Clinton st
Buckley, Charles F., 802 Carroll st
Buist, George Lamb, 90 Halsey st
Bunker, Henry A., 158 Sixth av
Burke, Frank T., 11 Halsey st

Burnett, Peter V., 170 Keap st
Butler, Chas. R., 584 Franklin av
Butler, Glentworth R., 229 Gates av
Butler, William E., 113 Halsey st
Callan, W. J., 192 Dean st
Callender, J. Meade, 172 Remsen st
Campbell, William F., 86 Greene av
Candidus, Eugene W., 215 Hewes st
Candidus, P., 228 S. Ninth st
Cardona, Lawrence J., 833a Lafayette av
Cardwell, John C., 553 Halsey st
Carney, James L., 243 Sixth av
Carpenter, F. E., 124 Reid av
Catlin, Arnold W., 207 Greene av
Chapman, William L., 19 Lafayette av
Chase, Carroll, 191 Kingston av
Chase, Walter B., 263 Hancock st
Chick, Edson Spaulding, 376 Gates av
Childs, Samuel B., 498 Classon av
Church, Stewart, 236 Clinton st
Clark, Charles Edward, 259 Lorimer st
Clark, Frank H., 758 Putnam av
Clark, Raymond, 310 Clinton av
Clark, Tracy Earl, 705 Macon st
Clarke, Albert Lincoln, 100 Nassau av
Clarke, Stanley G., 694 Halsey st
Clayland, J. M., 252 Rodney st
Cochran, Henry L., 141 Clinton st
Cochran, John A., 79 St. Marks av
Cohn, Michael A., 450 Stone av
Colby, George W., 142 Putnam av
Collins, Burnette C., 645 St. Marks av
Collins, John J., 1268 Bergen st
Colton, F. H., 136 Montague st
Combes, R. C. F., 185 Hancock st
Conkling, Henry, 143 Remsen st
Cook, Charles D., 162 Remsen st
Cook, Frederick A., 670 Bushwick av
Corcoran, Walter J., 233a Clinton st
Cornwall, Edward E., 146 Herkimer st
Coughlin, Robert E., 286 47th st
Coverly, J. H., 191 Washington park
Covey, John J., 287 Hoyt st
Cox, Charles N., 257 Jefferson av
Craig, Thomas C., 187 Washington park
Crawford, Frederick D., 441½ Clinton st
Cross., F. B., 131 Seventh av
Crowell, Edward P., 409 Jefferson av
Cruikshank, George H., 206 S. Oxford st
Cruikshank, William J., 102 Fort Greene pl
Curry, Albert M., 493 Classon av
Davis, G. H., 1016 Halsey st
Davis, John F., 400 Hancock st
Davis, William H., 289 Decatur st
Davidson, David, 445 State st
De Castro, Edward M., Jr., 482 Henry st
De Castro, Joseph F., 357a Clinton st
De Forest, Henry P., 369 Hancock st
De Long, William A., 17 Bainbridge st
De Lorme, Murette F., 340 Jay st
De Szigethy, Charles A. H., 157 Clinton st
De Waltoff, D., 297 47th st
Dee, William Vincent, 290 Bridge st
Deely, George E., 200 6th av
Delatour, H. Beeckman, 73 8th av
Dickert, John G., 928 Bushkick av
Dickinson, Robert L., 168 Clinton st
Dimock, Asa Redmond, 82 Sixth av
Dirkes, George J., 746 Driggs av
Dixon, Herbert S., 141 Lewis av
Dobkin, Nicholas, 190 Tompkins av
Dodge, Charles T., 651 Macon st
Dower, A. J., 380 Union st
Downey, James M., 381 Clinton st
Dreyer, Moritz Wolfson, 60 McKibben st
Drury, George, 235 Washington av
Dudley, William F., 32 Livingston st
Duffield, Warren L., 203 Sterling pl
Duncan, Cameron, 18 Clarkson st
Dunton, Harlow E., 355 President st
Duryea, Jesse T., 51 Center st., N. Y.
Dusseldorf, Louis M., 392 Union st
Eccles, Robert G., 191 Dean st
Edson, Benjamin, 83 St. Marks av
Edwards, Jessie S., 29 Spencer pl
Efstrom, Carl E., 437 Pacific st
Elliott, R. M., Long Island State Hospital
Eltinge, Edgar, 21 Macon st
Emerson, Florence G., 632 Bedford av
Emery, Z. Taylor, 481 Washington av
Engel, Walter F., 10 Stuyvesant av
Erdmann, A. F., 408 9th st
Essig, George, 488 Bedford av
Evans, George Alfred, 909 Bedford av
Everson, George, 364 Greene av
Fairbairn, Henry A., 249 McDonough st
Fedde, Bernard A., 773 Union st
Ferris, Edward D., 220 53rd st
Ferris, G. Newton, 910 Flatbush av
Fielding, G. B., 182 8th av
Figueira, Mathias, 14 Stuyvesant av
Finch, William Young, 407 Bergen st
Fincke, C. Louis 166 Clinton st
Fitzgerald, Frank J., 1081 Bergen st

Fitzsimmons, James C., 451 Gold st
Fleck, Louis H., 940 Bushwick av
Fleming, James W., 471 Bedford av
Fleming, Leon M., 100 Hanson pl
Foote, Louis N., 627 Marcy av
Foster, Clarendon A., 202 Berkeley pl
Fowler, C. B., 752 Carroll st
Fowler, George Ryerson, 301 DeKalb av
Fowler, Russell S., 301 DeKalb av
Franciscus, Henry, 80 Tompkins av
Fraser, Homer E., 18 S. Portland av
French, Thomas R., 150 Joralemon st
Frickenstein, Theo., 110 Union st
Friend, Walter M., 404 Clinton st
Fries, William A., 413 50th st
Frischbier, Otto Gustav, 235 Humboldt st
Fuhs, Jacob, 871 Park pl
Fuller, Frances V. C., 110 St. James pl
Gallagher, James T., 449 Lafayette av
Gardiner, Sidney H., 1085 Gates av
Gardiner, William F., 94 Prospect Park W.
Garnier, Anna M., 131 Milton st
Gartner, Charles, 259 Humboldt st
Gay, Frederick C., 106 Macon st
Geis, Norman P., 455 Classon av
Gifford, H. Lincoln, 294 Hooper st
Gildersleeve, Charles P., 18 Schermerhorn st
Gilfillan, William, 98 Remsen st
Gilfillan, William J., 79 Main st
Gilligan, Alexander, 962 Bedford av
Glynn, James P., 395 Ninth st
Glynn, John W., 71 1st pl
Golding, John F., 363 Franklin av
Goodrich, Charles H., 280 Park pl
Gordon, Onslow A., 666 Greene av
Gormly, John L. J., 430 Ocean Parkway
Grant, Walter S., 340 Stuyvesant av
Griffing, George P., 597 Lorimer st
Griffith, William A., 669 Willoughby av
Griffiths, Albert F., 604 E. 24th st
Gulick, Luther Halsey, Pratt Institute
Gunther, Eugene C., 184 5th av., Manh.
Gunther, Theodore C., 277 9th st
Hager, William C., 258 Hooper st
Hall, Gordon R., 164 Clinton st
Haller, J. Frederick, 484a McDonough st
Hamlin, Cyrus, 150 Putnam av
Hanan, James T., 322 Underhill av
Hancock, James Cole, 43 Cambridge pl
Happe, William H., St. John's Hosp., Brooklyn
Harcourt, Joseph M., 305 Clinton st
Harman, Eugene P., 563 St. Marks av
Harrigan, John, 401 Clinton st
Harrington, Burt D., 34 Lenox road
Harrison, Daniel A., 103 Montague st
Hatch, Edwin A., 823 DeKalb av
Hatton, J. Elizabeth, 508 Third st
Hawley, George R., 309 Gates av
Hegeman, Thomas B., 2600 Newkirk av
Hendrickson, Clifford W., 622 St. Marks av
Hendrickson, Skidmore, 1275 Bergen st
Henry, Charles C., 56 Clark st
Henry, J. Williams, 1383 Pacific st
Hepp, Charles F., 398 Graham av
Herkimer, Robert H., 181 Adelphi st
Herman, Charles T., 290 Halsey st
Herriman, F. R., 316 Quincy st
Hettesheimer, Charles J., 309 Wyckoff av
Hewett, W. B., 227 Clermont av
Hickok, Eugene P., 114 Pennsylvania av
Hicks, Edward E., 295 Stuyvesant av
Hicks, Elias R., 940 Flatbush av
Hill, John O. F., Neptune av cor. Cortland st
Hirsh, Charles H., 462 51st st
Hoag, David E., 985 Lafayette av
Hoar, Joseph D., 95 S. Portland av
Hodges, Edward, 156 Halsey st
Hoerle, August, 166 N. 6th st
Hoffman, Henry O., 138 Prospect park west
Hogan, Patrick F., 318 50th st
Holden, Frederick C., 63 7th av
Holley, I. McMunn, 636 St. Marks av
Holton, David C., 601 Leonard st
Hoole, Lester C., 77a Monroe st
Hoople, Heber N., 253 Gates av
Hopkins, George G., 350 Washington av
Horni, John, 179 Penn st
Hotchkiss, Henry T., 146 Halsey st
Howe, Alexander C., 311 Cumberland st
Hoxsie, Edward H., 1 Hart st
Hubbard, William S., 97 Halsey st
Hull, Thomas H., 55 Lee av
Hulse, Clarence H., 206 Monitor st
Humpstone, O. Paul, 92 Greene av
Hunt, Joseph H., 1085 Bedford av
Hussey, Augustus A., 409 Stuyvesant av
Hutchins, Alexander, 796 De Kalb av

Hutchinson, William M., 205 Clinton st
Hyde, Clarence R., 126 Joralemon st
Hyde, Joel W., 215 Schermerhorn st

Ingalls, James W., 874 Lafayette av
Ingram, Mary de B., 196 Joralemon st
Ives, Grace D., 422 Classon av
Ives, Robert F., 22nd av., cor 85th st

Jacobson, Arthur C., 115 Johnson st
Jamison, P. Chalmers, 139 Montague st
Jarrett, Arthur R., 95 Halsey st
Jenkins, Charles R., 31 Conselyea st
Jenkins, John A., 271 Jefferson av
Jenks, F. Percy, 419 9th st
Jennings, John E., 41 Halsey st
Jennings, William E., 931a Jefferson av
Jewett, Charles, 330 Clinton av
Jewett, Frederick A., 282 Hancock st
Jewett, Harold F., 609 Nostrand av
Jewett, William Averell, 382 Adelphi st
Joerg, Oswald, 12 Schermerhorn st
Johnson, J. G., 153 Joralemon st
Jones, Florence Leigh, 508 3rd st
Jones, Mortimer D., Kings Co. Hosp., Brooklyn
Judd, Albert M., 188 6th av
Junior, Kenneth F., 458 E. 29th st

Keenan, Henry C., 613 Willoughby av
Kene, Joseph A., 64 Greene av
Kennedy, Francis S., 462 Greene av
Kennedy, James C., 762 Willoughby av
Kenny, Eugene J., 219 Prospect pl
Kerr, C. Le Grand, 97 Cumberland st
Kessler, George L., 588 Bedford av
Ketterle, John, 221 Troutman st
Kevin, Christopher D., 719 Halsey st
Kevin, J. Richard, 252 Gates av
Keyes, Frank P., 77 Hanson pl
Keyes, James J., 226 17th st
Kidd, P. Edwin, 81 St. Marks av
King, James S., 146 McDonough st
King, Samuel T., 34 Greene av
Kinloch, Robert E., 283 Franklin av
Kinne, William, 46 Fourth av
Kirk, Frederick J., 404 Hamburg av
Klein, Valentine J., 1152 Lafayette av
Knause, B. Frank, 1094 Bushwick av
Knight, Frank H., 59 7th av
Koch, Alexander, 1135 Herkimer st
Koempel, Ludwig, 838 Bedford av
Koerner, William F., 154 Rodney st
Koethe, Hugo, 262 Vernon av
Kopf, Joseph B., 324 58th st
Kroos, Edward C., 151 Rodney st
Kuhn, George R., 122 Clinton av

Lack, C. Eugene, 692 10th st
Lamadrid, Julio J., 412 Greene av
Lane, William B., 395 Washington av
Lange, Hugo, 655 Bedford av
Langstaff, J. Elliott, 19 7th av
Langstaff, Lewis G., 175 6th av
Lawrence, Andrew W., 558 Bedford av
Lawrey, Nicholas, 84 S. Fourth st
Lee, John A., 366 Herkimer st
Le Fevre, Caroline H., 665 St. Marks av
Leiter, Joseph G., 616 McDonough st
Lester, John C., 179 Schermerhorn st
Levy, Max, 74 McKibben st
Lewis, Morris T., 414 55th st
Lewis, Stewart, 13 Cambridge pl
Leyh, George F., 71 Jefferson av
Liebermann, Gustave, 36 McKibben st
Lincoln, Harry Warren, 113 Hancock st
Linden, William, 640 Nostrand av
Linderoth, Martin, 50 Greene av
Lindredge, Edwin F., 157 Washington park
Lippelt, William J., 311 Melrose st
Little, Frank, 114 Montague st
Little, George F., 469 Clinton av
Little, William A., 923 Bedford av
Lloyd, T. Mortimer, 125 Pierrepont st
Longstreet, Arthur W., Norwegian Hospital
Louria, Leon, 255 Hewes st
Love, Cornelius R., 167 Clinton st
Lucas, David F., 552 Pacific st
Ludlum, Walter D., E. 16th st and av C.
Luhrsen, Ernest F., 336 Gates av
Lutz, Stephen H., 551 Madison st
Lynch, Eugene A., 661 Greene av
Lysaght, Ellen, 635 Willoughby av

McAlpin, William, 136 Concord st
McAveney, William J., 22 3rd st
McCabe, James H., 125 Williams st
McCammon, Frederick J., 359 Stuyvesant av
McCleary, Thomas F., 915 Bedford av
McClelland, Lefferts A., 78 McDonough st
McConville, C. Adeline, 195 Lorimer st
McCorkle, John A., 149 Clinton st
McDonough, Fergus, 29 Poplar st
McEvitt, James, 45 3rd pl
McEvitt, John C., 407 Clinton st

McFarlane, Thomas J., 206 36th st
McGuire, Constantine F., 503 Clinton st
McLean, Henry C., 146 6th av
McLeod, John A., 154 Noble st
McNamara, Sylvester J., 325 Union st
McNaughton, George, 479 Clinton av
McPhail, Leonard C., 161 Hicks st
McQuillan, John P., 250 58th st
McVean, Charles H., Kings Co. Hosp.
MacCoy, Cecil, 151 Clinton st
MacGillvary, Stanley H., 820 Bedford av
Macumber, John T., 209 Greene av
Maddock, George F., 80 McDonough st
Maddren, William, 1 Hanson pl
Maddren, William Harvey, 131 S. Oxford st
Magilligan, Francis J., 135 Bergen st
Mahr, George J., 88 Wilson st
Malament, Manasseh J., 68 McKibben st
Malone, Joseph W., 828 22nd av
Manecke, Phillip G., 1292 Bushwick av
Mangan, Daniel C., 95 Park av
Manley, Mark, 261 Monroe st
Marsh, Edward F., 466 9th st
Marshall, Joseph H., 506 Fulton st
Martin, W. Ross, 724 Greene av
Mason, Lewis D., 171 Joralemon st
Matheson, A. Ross, 37 7th av
Matheson, Sewell, 578 Bergen st
Mathewson, Arthur, 139 Montague st
Matson, Nathaniel, 415 Greene av
Mayne, Earle H., 8744 18th st
Mead, Raphael M., 381 Union st
Medd, John C., 141 Seventh av
Merolla, Genaro, Ocean Parkway, bet. avs B and C
Merzback, Joseph, 446 Pacific st
Meury, John B., 785 Bushwick av
Meyer, David W., 161 Clinton st
Meyer, Joseph, 228 Vernon av
Meyersburg, Adolph G., 102 Bradford st
Midgley, Joseph E., 1125 Dean st
Miller, Francis H., 894 St. John's pl
Miller, Maud, 133 Milton st
Miller, Franklin P., 314 Stuyvesant av
Miller, Lewis H., 107 Halsey st
Millington, William F., 356 9th st
Mills, Henry M., 192a 6th av
Moitrier, William, 119 Lorimer st
Molin, Charles G., 184 State st
Mooney, P. J., 118 Kent st
Moore, Julius H., 282 Halsey st
Morgan, James F., 14 Spencer pl
Morris, Edward J., 44th st near 3rd av
Morrison, Robert J., 354 Tompkins av
Morton, Henry H., 32 Schermerhorn st
Morton, Lawrence J., 303 Henry st
Mosher, Burr B., 44 Court st., Temple Bar
Mosher, Eliza M., 44 Court st., Temple Bar
Moxam, P. W. T., 604 E. 24th st
Mucklow, Albert E., 300 Sumner av
Mueller, Carl, 9 Lewis av
Munson, Forbes J., 330a Lafayette av
Muren, G. Morgan, 73 Orange st
Murphy, Joseph P., 620 St. Marks av
Murray, Archibald, 69 Remsen st
Myerle, David, 510 Bedford av
Myers, William A., 1411 Bushwick av
Mykrantz, Howard B., 37 Decatur st
Mylod, Thomas F., 566 Washington av
Napier, Charles Dwight, 1273 Bedford av
Nehrbas, Jacob, 1368 Myrtle av
Neiman, L. Allen, 73 Henry st
Neuss, William, 82 Stanhope st
Newman, Frank R., 167 Hancock st
Nichol, Harry W., 323 Sterling pl
Nichols, Louis L., 386 Stuyvesant av
Nickerson, Lorenzo M., 89 S. 9th st
Nielson, John C. E., 753a Union st
North, Nelson L., 627 Bedford av
North, Nelson L., Jr., 118 Hooper st
Northridge, Augustus P., 121 Convert st
Northridge, Thomas H., 320 Cumberberland st
Northridge, William A., 21 Hanson pl
Oatman, Edward L., 82 Remsen st
O'Neill, John H., 897 Kent av
Onuf, B., 99 Berkeley pl
Ormiston, Robert, 117 S. Elliott pl
Ostrander, George A., 61 Greene av
Otis, F. Burton, 338 Halsey st
Paffard, Frederick C., 238 Clinton st
Page, Emmett D., 304 Washington av
Paine, Arthur R., 211 Lafayette av
Palmer, Ernest, 155 Clinton st
Parker, Dewitt L., 167 Remsen st
Parker, Edward A., 262 McDonough st
Parrish, John W., 11 Montague st
Parrish, Paul L., 1448 Bedford av
Pascual, William V., 607 St. Marks av
Peacock, John M., 249 Jefferson av
Pearce, Eugene F., 95 Henry st
Pearson, Lewis W., 401 Union st

Peck, Herman T., 321 Halsey st
Peebles, Martha J., 29 Spencer pl
Pell, Arthur, 1149 Dean st
Pendleton, Judson P., 42 Seventh av
Pennington, William J., 184 S. 9th st
Peterman, Charles P., 809a Greene av
Petersen, Christian E., 1067 Dean st
Pettit, H. S., Adelphi College
Pettit, Steven C., Neck Rd., Gravesend, L. I.
Philleo, Willis H., 155 Herkimer st
Phillips, Charles A., 371 Greene av
Pierce, George H., 76 St. James pl
Pierce, John W., West 8th st., Coney Island
Pilcher, Lewis S., 386 Grand av
Pineo, Thomas A., 402 McDonough st
Polak, John O., 287 Clinton st
Pomeroy, Ralph H., 511 Nostrand av
Pool, Charles S., 194 Schermerhorn st
Pool, William P., 147 Clinton st
Porter, E. Pender, 1 Glenada pl
Pratt, William H., 94 6th av
Prendergast, Patrick J., 340 Clinton st
Price, Henry R., 163 Hancock st
Price, Walter D., 653 St. Mark's av
Prout, Jonathan S., 26 Schermerhorn st
Pullman, James, 155 Reid av
Pyburn, Paul F., 600 Franklin av
Pyles, Marion, 47 Hancock st
Quinn, John R., 314 Greene av
Rae, Alexander, 117 Henry st
Randall, Edward T., 139 Hancock st
Rankin, John, 600 Jefferson av
Rankin, William H., 151 Hancock st
Rappold, Julius C., 760 Bushwick av
Rathburn, Nathan P., 201 Greene av
Raub, Joseph M., 295 Clinton st
Raymond, Joseph H., Polhemus Mem. Clinic
Raynor, Addison, 977 Flatbush av
Read, Henry N., 228 Clinton st
Reed, George E., 737 Putnam av
Reque, Peter A., 551 Henry st
Reynolds, Edwin, 129 Lafayette av
Reynolds, Willard G., 162 Halsey st
Rickard, Wilbur L., 262 Stuyvesant av
Riegelman, Laura M., 43 Lee av
Riggs, Herman C., 117 Montague st
Risch, Henry F., 480 14th st
Risch, Otto E., 495 3rd st
Ritter, August H., 262 Hewes st
Roberts, Dudley P., 165 Clinton st
Robertson, Victor A., 834 Union st
Rogers, Herbert C., 377 Gates av
Rogers, Hugh E., 36 Troutman st
Rooney, Alexander, 230 17th st
Ross, Frank H., 278 Bridge st
Ross, Walter H., 185 Hewes st
Royce, Rubert S., 211 Greene av
Russell, Julien W., D.D.S., 374 Adelphi st
Rutz, Anthony A., 200 Sixth av
Santoire, Henry A., 148 Clinton st
Santoire, Samuel, 148 Clinton st
Sauer, C. Theodore, 284 Sixth av
Schaffner, Phillip M., 262 Vernon av
Schalck, Ernest, 426 Quincy st
Schauf, Adam, 198 Vernon av
Schelling, Henry L., 841 Willoughby av
Schenck, Peter L., 95 6th av
Schirmer, Emilie C., 574 Franklin av
Schirmer, William C., 574 Franklin av
Schlatter, Bernard C., 158 17th st
Schlitz, Francis A., 28 Jefferson av
Schmidt, John A. D., D.D.S., 1195 Dean st
Schmidt, William C. J., 21 Bleecker st
Schoenijahn, W. Carl, 822 Union st
Schroeder, William, 339 President st
Schroeder, William, Jr., 513 11th st
Schwab, Alvin H., 313 McDonough st
Scofield, Charles E., 152 Taylor st
Scott, Peter, 128 Reid av
Scoville, William J., Church st., Richmond Hill
Scrimgeour, Robert, 752 Carroll st
Search, Charles J., 168 Putnam av
Seeley, Howard J., 151 Stuyvesant av
Seimal, William A., 275 Lorimer st
Senior, Frank S., 248 Arlington av
Seymour, Wilbur H., 233 Clinton st
Sharpe, Frank M., 125 So. Oxford st
Shattuck, Warren S., 147 Clinton st
Shaw, Frank W., 327 Greene av
Shaw, Richard E., 115 Amity st
Shea, J. Denton, 427 8th st
Shepard, A. Warner, 126 Willoughby st
Shepard, Charles H., 81 Columbia Heights
Shepard, William H., 202 52nd st
Sheppard, John E., 130 Montague st
Sherman, Wesley, 336 Ninth st
Sherwell, Samuel, 33 Schermerhorn st
Sherwood, Walter A., Bergen st and Carlton av
Shipley, Alfred E., 239 Lincoln pl
Shoop, Frederick J., 316 Cumberland st
Showers, Harriet D. W., 184 17th st
Siegel, F., 82 Lee av
Simmons, Warren S., 338 Lafayette av
Simmons, William, 23 Schermerhorn st

Simpson, John F., 2 Broome st
Simrel, George W., 285 Clarkson st
Slee, Arthur W., 1230 Hancock st
Slote, Samuel H., 75 McKibben st
Smith, Henry M., 64 Montague st
Smith, Joseph E., 92 Lee av
Smith, J. Wheeler, 1120 Herkimer st
Smylie, Arthur E., 132 Penn st
Snyder, William H., 141 Montague st
Somers, James A., 32 Lafayette av
Sparks, Agnes, 140 S. Portland av
Spence, Thomas B., 139 Seventh av
Staebler, David M., 690 Macon st
Steinhe, C. O. Herman, 220 17th st
Sterling, John H., 225 Schermerhorn st
Stern, Bernard, 633 E. 28th st
Stevenson, Robert, 99 India st
Stickle, Charles W., 1315 52nd st
Stivers, George L., 303 Vanderbilt av
Stivers, John R., 180 Lefferts pl
Stoney, F. E. A., 229 82nd st
Stratton, William E., 195 Berkeley pl
Straub, George E., 385 Franklin av
Stuart, Francis H., 123 Joralemon st
Sturges, Purdy H., 145 Seventh av
Sturgis, Elizabeth M., 131 Milton st
Sullivan, John D., 74 McDonough st
Sullivan, Richard H., 42 Macon st
Sullivan, William E., 532a Henry st
Swalm, William F., 118 Lafayette av
Swan, Eugene L., 418 Lafayette av
Tag, Charles H., 168 Keap st
Tallmadge, Andrew T., 2118 Albemarle Rd
Taylor, John M., 438 3rd st
Taylor, J. Richard, 1275 Bedford av
Taylor, Stephen L., 558 Madison st
Taylor, Vernon E., 104 Utica av
Taylor, William Henry, 714 Lafayette av
Terhune, James J., 169 Adelphi st
Terry, Charles H., 540 Washington av
Thomson, George G., 89 Sterling pl
Tieck, Gustav J. E., 89 Pulaski st
Tieste, Louis E., 6 Lafayette av
Todd, Joseph F., 550 Carlton av
Tomes, William Austin, 500 Classon av
Townsend, Palmer, 588 Jefferson av
Treadwell, George H., 64 S. Portland av
Tredwell, Alonzo S., 107 McDonough st
Truex, Stephen P., 257a Madison st
Truslow, Walter, 142 Clinton st
Tucker, Harrison A., 393 Clinton st
Tupper, Charles O., 329 Jefferson av
Turner, Henry C., 412a Clinton st
Turner, Leslie A., 256 Hancock st
Turner, William J., 105 Clinton st
Tuthill, Henry R., 83 Monroe st
Tuthill, James Y., 100 Fort Greene pl
Van Cott, Joshua M., 188 Henry st
Vietor, Edward W., 166 St. James pl
Wackerhagen, George, 28 7th av
Wade, H. Albert, 495 Greene av
Wadsworth, Emory M., 60 Brooklyn av
Waldie, John L., 258 McDonough st
Wallace, Henry, 183 Congress st
Walsh, John E., 633 Tenth st
Warbasse, James P., 68 Greene av
Wardenburg, George J., 144 St. Mark's av
Warner, Horace S., 319 McDonough st
Warren, D. Edward, Long Island State Hospital
Waterman, James S., 1193 Dean st
Waterworth, William, 3 Hancock st
Watt, James, 174 Clinton st
Waugh, Darwin W., 388 Clinton st
Webster, Henry G., 162 Halsey st
Weed, Ver Nooy W., 675 Jefferson av
Weisbrod, Frederick, 882 Bushwick av
Wells, Joseph E., 410 Clinton st
Wells, Thomas L., 945 St. Mark's av
Welton, R. Bradlee, 810 Union st
Wenzel, Henry G., 387 Ralph av
West, Frank E., 172 Clinton st
Westbrook, George R., 175 Schermerhorn st
Westbrook, Richard W., 1145 Dean st
Weygandt, Frederick G., 645 Bedford av
Wheatley, Tenny H., 151 Hewes st
Wheeler, Edward A., 110 Ross st
Wheeler, Robert T., 210 Lee av
Wight, J. Sherman, 30 Schermerhorn st
Wilgus, Sidney D., Kings Co. Hospital
Williams, George A., 449 Hancock st
Williams, Herbert F., 197 Gates av
Williams, John G., 753a Union st
Williams, Ralph C., 317 51st st
Williamson, Charles E., 211 Van Buren st
Williamson, Charles S., 491 Greene av
Willis, Royal H., 394 Jefferson av
Wilson, E. E., 120 Brooklyn av
Wilson, Ezra H., 194 Keap st
Wilson, Frank E., 1242 Bushwick av
Winfield, James McFarlane, 47 Halsey st
Winter, Henry L., Cornwall, N. Y.
Winter, Frederick G., 18 Patchen av
Wood, Frederick J., 2 St. James' pl

Wood, J. Scott, 172 6th av
Wood, Walter C., 1276 Pacific st
Woolsey, William C., 29 Lafayette av
Wright, Edward W., 115 Montague st
Wunderlich, Frederick W., 165 Remsen st
Yerdon, Charles F., 1276 Herkimer st
Zabriskie, Edward G., 878 Flatbush av
Zabriskie, John B., 2103 Church av
Zahn, Anthony F., 115 Sumner av
Zellhoefer, Charles, 175 S. 4th st
Zimmerman, Victor L., 557 Halsey st
Number of Members, 707.

MEDICAL SOCIETY OF THE COUNTY OF LEWIS.

(Organized January 8, 1861.)

MEETINGS.—Annual, first Tuesday in June; semi-annual, first Tuesday in December.

Officers. (1902.)

C. P. Kirley, *President.*
W. O. Hubbard, *Secretary.*
J. H. Watson, *Vice-President.*
J. Z. Hunt, *Treasurer.*

Members.

F. D. Bigarel, Port Leyden
W. H. Cramer, Copenhagen
F. A. Crane, Lowville
O. G. Harrington, Constableville
W. O. Hubbard, Lowville
James Z. Hunt, Lowville
W. H. Johnston, Port Leyden
F. E. Jones, Beaver Falls
C. P. Kirley, Lowville
A. C. Miller, Turin
S. H. Murphy, Glenfield
P. H. Von Zierolshofen, Croghan
J. H. Watson, Turin
Number of Members, 13.

MEDICAL SOCIETY OF THE COUNTY OF LIVINGSTON.

(Organized June, 1806.)

MEETINGS.—Annual, second Tuesday in June; semi-annual, second Tuesday in December.

Officers. (1903.)

William Spratling, *President.*
George W. Squires, *Secretary.*
F. E. Strasenburgh, *Vice-President.*
G. G. Jones, *Treasurer.*

Censors: J. P. Brown, F. J. Bower, C. H. Richmond, W. E. Lauderdale, R. J. Menzie,

Members.

Irving C. Allen, Avon
F. J. Bowen, Mt. Morris
John P. Brown, Nunda
L. W. Byam, Mumford
J. E. Cresfield, Dansville
F. B. Dodge, Mt. Morris
Fred. R. Drisebach, Dansville
J. A. M. Dike, York
Robert W. Green, Geneseo
Jacob M. Hagey, Mt. Morris
Edward L. Hanes, Sonyea
George G. Jones, Geneseo
George H. Jones, Fowlerville
W. E. Lauderdale, Geneseo
W. K. McGowan, Conesus
R. J. Menzie, Caledonia
Wm. H. Montgomery, Sonyea
J. A. Morrissey, Lima
F. H. Moyer, Moscow
William Nisbet, Avon
B. Onuf, Sonyea
F. M. Perine, Dansville
Edward C. Perry, Avon
John C. Preston, Avon
Charles H. Richmond, Livonia
Wm. T. Shanahan, Sonyea
Edward A. Sharp, Sonyea
William P. Spratling, Sonyea
Frederick E. Squires, Livonia
George W. Squires, East Avon
F. A. Strasenburgh, Avon
Annie M. Tremaine, Sonyea
Fred. A. Wicker, Hemlock Lake
Number of Members, 33.

MEDICAL SOCIETY OF THE COUNTY OF MADISON
(Organized July 29, 1806.)

MEETINGS.—Annual, second Tuesday in May.

Officers. (1903.)

N. O. Brooks, *President.* M. R. Joy, *Vice-President.*
E. H. Carpenter, *Secretary.* H. G. Germer, *Treasurer.*
Censors: C. H. Perry, E. P. Bailey,
O. S. Langworthy, D. D. Chase.

Members.

R. H. Ash, Canastota
E. P. Bailey, Oneida
L. C. Beebe, Hamilton
M. Billington, Chittenango
Gilbert Birdsall, North Brookfield
N. O. Brooks, Oneida
H. Clift Brown, Brookfield
O. W. Burhyte, Brookfield
E. H. Carpenter, Oneida
H. W. Carpenter, Oneida
M. Cavana, Oneida
D. D. Chase, Morrisville
C. M. Colegrove, Canastota
G. W. Davis, Peterboro
L. R. Davis, Oneida
G. G. Decker, West Eaton
F. E. Dewey, Peterboro
A. P. Dodge, Oneida
W. H. Douglas, East Hamilton
F. C. Drake, Oneida
J. R. Eaton, Chittenango
Herman G. Germer, Canastota
W. H. Griffith, Munnsville
A. N. Haskins, Hubbardsville
James F. Huntley, Oneida
M. R. Joy, Cazenovia
J. W. Knapp, Canastota
O. S. Langworthy, Hamilton
J. E. McClelland, De Ruyter
G. W. Miles, Oneida
Edgar L. Miller, Eaton
S. P. Moore, Munnsville
C. H. Perry, Oneida
Otto Pfaff, Oneida
A. D. Smith, New Woodstock
H. H. Stone, Oneida
William Taylor, Canastota
H. H. White, Earlville
S. J. Wilson, Oneida

Number of Members, 39.

Deceased: I. N. Goff, Cazenovia, October 13, 1902, æt. 69; H. P. Mead, Morrisville, May 24, 1902, æt. 72.

MEDICAL SOCIETY OF THE COUNTY OF MONROE.

MEETINGS.—Annual, last Wednesday in May.

Officers. (May, 1903.)

Wheelock Rider, *President.* William M. Brown, *Vice-President.*
Wm. L. Conklin, *Secretary.* C. A. Greenleaf, *Treasurer.*

Members.

(Where no town is mentioned Rochester is to be understood.)

John H. Acheson, 115 Atlantic av
M. May Allen, 297 Alexander st
J. A. Ames, East Rush
F. D. Andrew, 28 S. Union
L. B. Andrews, 560 East Main st
E. B. Angell, 295 Alexander st
Ogden Backus, 5 Granger pl
Samuel B. Baker, 185 Wilder st
Evylin P. Baldwin, 476 West av
E. P. Ballantine, State Hospital
C. R. Barber, 52 Cumberland st
W. M. Barron, 34 N. Fitzhugh st
W. D. Becker, 8 Franklin sq
George E. Beilby, State In. School
A. W. Bellamy, 202 E. Main st
G. C. Benjamin
J. S. Berkman, 348 N. Clinton av
Perry A. Bly, 98 Park av
H. W. Bode, 323 Joseph av
E. C. Boddy, 19 Jones av
C. O. Boswell, 389 East av
J. Brady, 397 Plymouth av
C. B. Braman, 394 Joseph av
John D. Briggs, Williamson
C. M. Briggs, Fairport
W. M. Brown, Brighton
M. Alice Brownell, Newark
Kathleen L. Buck, 279 Clinton av
James C. Buckley, 10 Cumberland st
W. H. Bullis, 234 University av

J. J. A. Burke, 65 East av
George S. Burns, 684 Clinton av No.
Ina V. Burt, Phelps
Joseph S. Campbell, 41 Clinton av So.
P. D. Carpenter, Pittsford
George G. Carroll, 304 West av
R. L. Carson, 105 East av
George G. Carwell, Jr., 304 West av
R. C. Cartwright, 82 Ambrose st
J. W. Casey, 137 Plymouth av
M. L. Casey, 137 Plymouth av
I. Taylor Clark, 15 Fulton av
W. B. Cochrane, Brighton
Philip J. Conboy, 73 Clinton av No.
J. B. Cowles, Fairport
Morey S. Collier, 1 Athens st
Charles G. Combs, 185 Monroe av
C. V. C. Comfort, 38 Savannah st
W. L. Conklin, 720 South av
William B. Connors, 381 Andrew st
D. J. Corrigan, West Webster
J. A. Cormier, 355 Central av
Robert G. Cook, 242 Alexander st
H. H. Covel, 105 East av
Charles S. Craig, Hamlin
James F. Crowley, 309 E. & B. Bldg
Rolin O. Crosier, Monroe Co. Alms House
J. R. Culkin, 213 Frank st
D. F. Curtis, 89 South av
A. Dann, 406 Granite Building
C. E. Darrow, 116 East av
James C. Davis, 8 Chestnut st
C. A. Dewey, 53 S. Fitzhugh st
M. E. Dickinson, 604 Powers Bldg
Sarah R. A. Dolly, 52 East av
F. F. Dow, 137 Park av
H. S. Durand, 87 S. Fitzhugh st
Edgar H. Earl, 420 Lyell av
Frederick East, 260 North st
W. S. Ely, 78 S. Fitzhugh st
S. L. Elsner, 83 St. Paul st
G. A. Engert, 43 Clinton av South
John J. Evans, 123 Frank st
W. V. Ewers, 140 N. Goodman st
Porter Farley, 801 St. Paul st
H. M. Fenno, 34 Hamilton st
Jas. H. Finnessy, 375 Clinton st No.
J. B. Finnecane, Mendon
G. T. Fischer, 54 Clinton av
G. W. Foster, 19 East av
William F. Frasch
A. B. Frazee, 61 Clifford st
E. A. French, 505 North st
George P. French, 71 Prospect st
Robert T. French, 209 Alexander st
W. S. Fuller, Fairport
James E. Gage, 525 Fitzhugh st
L. T. Gandy, Chili
Horace Gee, 597 Lake av
G. B. Gilbert, 11 Meigs st
F. H. Goddard, 265 Meigs st
George Goler, 127 East av
H. C. W. Graham, 69 East av
Erlo H. Gray, 4 Atlantic av
C. A. Greenleaf, 209 East av
O. Groves, 299 Central Park
P. C. Guinan, 1 Cataract st
A. R. Gumberts, 48 Chatham st
E. M. Hague, 650 Main st E.
W. S. Hall, 293 Monroe av
Eugene F. Hamburg, Genesee st near Brooks av
E. R. Hardenbrook, 251 Lake av
E. A. C. Harris, 400 Lake av
I. E. Harris, 400 Lake av
D. G. Hastings, 272 Alexander st
Sumner Hayward, 84 East av
John H. Hazen, Brockport
A. W. Henckell, 96 Sophia st
W. J. Herriman, 263 West av
Edw. A. Hoffman, 283 Central Park
S. A. Holman, Hilton
B. L. Hovey, 34 N. Fitzhugh st
E. H. Howard, Rochester State Hosp.
W. R. Howard, 660 Main st E.
W. J. Howe, Scottsville
H. B. Howell, 228 Hudson av
Loren W. Howk, 370 West av
C. A. Huber, 51 Monroe av
De Witt C. Hunter, 607 St. Paul st
J. M. Ingersoll, 275 Monroe st
Thomas Jameson, 240 West av
A. M. Johnson, 289 Main st E.
Ira T. Johnson, 138 Fulton av
Frank A. Jones, 309 Lake av
S. C. Jones, 207 East av
Wm. B. Jones, 525 Lake av
O. E. Jones, 392 Main st E.
Joel G. Justin, 20 Gibbs
H. M. Justin, 20 Gibbs
J. G. Keeler, 19 Vick Park A.
J. J. Kempe, Florida
N. F. Kiefer, 663 Clinton av No.
Thos. A. Killip, 462 Clinton av No.
H. Koch, 19 Cumberland st
W. G. Lacy, Scottsville
George A. Lane, 303 University av
E. H. Lapp, Williamson
C. T. La Moure, Rochester State Hospital
Howard A. La Moure, Rochester State Hospital
Frank P. Leadley, 394 West av
M. E. Leary, 397 West av
H. J. Levis, 185 Fulton av
S. H. Linn, 243 Alexander st
David Little, 162 Plymouth av
S. W. Little, 162 Plymouth av
M. L. Lord, Oxford st
C. H. Losey, 145 Lake av
M. C. Lyons, Ithaca

J. W. McCauley, 349 Monroe av
N. D. McDowell, 19 East av
J. T. McGoveron, 67 Clinton av So.
F. B. McKenney, 39 Lake av
Thos. F. McNamara, 446 Genesee st
J. W. Magill, 3 Park av Fairport
A. P. Maine, Webster
F. W. Maloney, 332 West av
F. A. Mandeville, 63 East av
H. J. Mann, Brockport
W. B. Mann, Brockport
G. A. C. Mannell, 38 Clinton av So.
G. A. Marion, 474 N. Goodman st
D. G. Mason, East Henrietta
M. C. Mason, 114½ Ambrose st
F. B. Maynard, 38 Clinton av So.
Louis K. Mezger, 193 Clinton av No.
E. M. Moore, Jr., 74 S. Fitzhugh st
R. M. Moore, 74 S. Fitzhugh st
T. T. Mooney, 116 Sophia st
E. W. Mulligan, 290 West av
W. T. Mulligan, 290 West av
P. H. Murphy, 117 Lake av
J. F. Mulherin, Syracuse
H. R. Nettleton, 270 Lake av
E. G. Nugent, 78 S. Fitzhugh st
Francis J. O'Brien, 441 Lake av
T. A. O'Hare, 97 State st
E. S. Olin, 178 Andrews st
I. D. Ozmun, 35 Clinton av So.
John E. Ottaway, Charlotte
M. B. Palmer, 89 Clinton av No.
E. G. Paxton, 113 North st
Joseph Pease, Hamlin
S. Perry, 103 S. Fitzhugh st
H. C. Phillips, 166 Frank st
S. A. Pierce, 422 Monroe av
A. S. Pike, 303 Alexander st
Ida May Porter, 347 Monroe av
E. B. Potter, State Hospital
M. C. Potter, 18 Gibbs st
Ezra B. Pratt, Fairport
B. I. Preston, Naples
J. Ready, 20 Gibbs st
Charles Reitz, 149 Hudson av
A. C. Remington, 275 West av
Frederic Remington, 500 Powers Bldg
A. L. Richmond, Morton
C. E. Rider, 53 S. Fitzhugh st
Wheelock Rider, 53 S. Fitzhugh st
Levi L. Riggin, Chili av
A. S. Rockwell, 46 Elizabeth st
John O. Roe, 28 Clinton av No.
Joseph Roby, 129 S. Fitzhugh st
L. W. Rose, 301 Alexander st
J. L. Roseboom, 672 Main st E.
Moses Rosenberg, 308 Central av
S. H. Rosenthal, 83 St. Paul st
E. W. Ruggles, 294 Alexander st
T. D. Rupert, Geneva
Martin Rutherford, 117 Campbell st
H. L. St. John, 116 N. Goodman st
F. H. Sawers, 548 Lake av
H. Schoonmaker, Seneca Falls
Justin Schopp, Clinton av No.
Q. C. Schuhart, 484 Clinton av No.
H. P. Sheldon, 139 Glenwood av
J. T. Sherman, 507 North st
Mary Hadley Smith, 72 East av
Mary J. Slaight, 79 Chestnut st
L. E. Slayton, Spencerport
Albert C. Snell, 138 S. Fitzhugh st
N. W. Soble, 381 Andrews st
L. J. Somers, 52 Clinton st So.
O. T. Stacy, 284 Alexander st
Wm. Stanton, Webster
J. A. Stapleton, 74 East av
Mary E. Stark, 35 Chestnut st
D. Starkey, Chili
C. S. Starr, 95 North st
W. G. Stedman, 94 East av
Peter Stockschlader, 646 South av
E. V. Stoddard, 68 S. Washington st
R. L. Stoddard, 38 Clinton av So.
J. H. Sullivan, Charlotte
Wm. H. Sutherland, 53 S. Fitzhugh st
J. E. Sutton, Scottsville
T. O. Tait, 374 Andrews st
M. M. Taplin, 77 Monroe av
D. C. Throop, 145 North av
Harriet M. Turner, 275 Alexander st
A. W. Thomas, 490 Lydell av
C. B. W. Thomas, 490 Lydell av
George P. Thomas, 3 Lake View Pk
Charles J. Tucker
F. J. Tunmore, 363 St. Paul st
Loretta W. Turner, 16 Chili av
P. G. Udell, Spencerport
J. C. Urquhart, 56 North st
C. A. Vanderbeck, 16 Gibbs st
M. D. Van Horn, Churchville
L. Allen Walker, 343 West av
George Waldron, 408 Plymouth st
Wm. T. Wallace, 354 Central av
W. Douglas Ward, 12 Grove pl
John E. Weaver, 385 Monroe av
Wm. H. Webb, 14 Windsor st
L. A. Weigel, 209 East av
Richard K. Wheeler, 210 Hudson st
J. W. Whitbeck, 322 East av
Charles E. White
V. A. Wickens, 289 West av
G. H. White, 464½ Plymouth av
C. W. Wilbor, 763 St. Paul st
I. T. Wile, 381 Andrew st
H. T. Williams, 274 Alexander st
W. W. Williams, Hilton
Benjamin Wilson, 101 Lake av
W. D. Wollf, 62 Cumberland st
C. D. Wooden, 129 Frank st

Lettie H. Woodruff, 17 Tremont st
Charles D. Young, 5 Gibb st
F. W. Zimmer, 45 Monroe av
John Zimmer, 53 Cumberland st

Number of Members, 272.

Deceased: Azel Backus, Rochester, September 2, 1902, æt. 74; James Beahan, Rochester; A. N. Braham, Brockport; Charles McD. Cameron, Rochester; John D. Dunning, October 27, 1901, æt. 76; H. B. Miner, West Mendon, January 2, 1893, æt. 96; E. M. Moore, Rochester, March 3, 1902, æt. 89; E. E. Whitcomb, Rochester, August 1, 1901, æt. 50.

MEDICAL SOCIETY OF THE COUNTY OF MONTGOMERY.

(Organized July, 1806.)

MEETINGS.—Annual, second Wednesday in December, at Fonda; semi-annual, second Wednesday in June, at Canajoharie; quarterly, second Wednesday in September and March, at Amsterdam.

Officers. (December, 1902.)

James B. Conant, *President.*
Otis Z. Bouton, *Secretary.*
F. I. Jansen, *Vice-President.*
W. J. Peddie, *Treasurer.*

Members.

Douglas Ayres, Fort Plain
Otis Z. Bouton, Fultonville
E. F. Bronk, Amsterdam
F. V. Brownell, Canajoharie
I. I. Buckbee, Fonda
E. J. Collier, Amsterdam
James B. Conant, Amsterdam
C. E. Congdon, Fort Plain
C. W. De Baun, Fonda
Wm. De Lamater, Minaville
James R. Fairbanks, Amsterdam
A. B. Foster, Fonda
L. A. Frazier, Amsterdam
S. H. French, Amsterdam
H. L. Furbeck, St. Johnsville
Archibald Gilbert, Amsterdam
Horace M. Hicks, Amsterdam
F. A. Husted, Amsterdam
Thomas G. Hyland, Amsterdam
F. I. Jansen, Fonda
R. G. Johnson, Amsterdam
Charles M. Klock, St. Johnsville
John W. Kniskern, Amsterdam
Edgar B. Lake, Amsterdam
Sherwood Le Fevre, Amsterdam
Daniel M. MacMartin, Amsterdam
George L. Meyer, Stone Arabia
H. W. Murphy, Amsterdam
Wm. J. Peddie, Fultonville
Wm. R. Pierce, Amsterdam
Julia K. Ina, Amsterdam
E. E. Rulison, Amsterdam
Julius Schiller, Amsterdam
Clarence Sicard, Amsterdam
F. E. Simons, Canajoharie
A. V. H. Smythe, Amsterdam
Charles Stover, Amsterdam
P. L. Suits, Tribes Hill
N. F. Sweatman, Amsterdam
D. M. Taylor, Amsterdam
C. F. Timmerman, Amsterdam
C. C. Vedder, St. Johnsville
Fred D. Vickers, Canajoharie
J. S. Walton, Amsterdam
S. A. Wessels, Canajoharie
Wm. M. White, Amsterdam

Number of Members, 46.

MEDICAL SOCIETY OF THE COUNTY OF NEW YORK.

MEETINGS.—Annual, fourth Monday in October; stated meetings, the fourth Monday in each month, June, July, and August excepted, at the New York Academy of Medicine, 17 West Forty-third Street.

Officers. (1903.)

Charles N. Dowd, *President.*
R. G. Freeman, *2d Vice-President.*
John Van Doren Young, *Secretary.*
Wendell C. Phillips, *1st Vice-President.*
John S. Warren, *Treasurer.*
Edmund P. Fowler, *Asst. Secretary.*

Censors: Frank Van Fleet, *Chairman;* H. S. Stearns, *Secretary;* William H. Park, Floyd M. Crandall, Joseph Brettauer.

Counsel for the Society: Hill, Stürcke & Andrews, 52 William Street.

Editor of Medical Directory: Edmund Prince Fowler.

Active Members.

Abbe, Robert, 13 W. 50th st
Abrahamson, Isador, 134 W. 126th st
Acken, Thomas Moore, 267 W. 45th st
Adams, Calvin Thayer, 6 E. 34th st
Adams, Charles Francis, 104 W. 73d st
Adams, John L., 38 E. 51st st
Adams, Robert Staunton, 6 E. 34th st
Addy, Arthur Rockwell, 500 W. 143d st
Adler, Isaac, 22 E. 62d st
Adriance, Vanderpoel, 105 E. 39th st
Agan, David H., 1074 Lexington av
Agatston, Sigmund A., 50 W. 112th st
Aitken, J. Francis, 218 E. 35th st
Aldrich, John, 164 W. 81st st
Alexander, David Esterbrook, 22 W. 115th st
Alexander, Isidor H., 218 E. 12th st
Alexander, Samuel, 5 W. 58th st
Alexander, Welcome T., St. Nicholas av and W. 157th st
Allen, Charles Lewis, State Hospital, Trenton, N. J.
Allen, Charles W., 30 E. 33d st
Allen, Thomas H., 180 W. 59th st
Allison, Hendery, 260 W. 57th st
Altman, Emil, 748 5th av
Altman, Max, 121 E. 113th st
Altshul, Henry, 153 W. 76th st
Alvarez, J. R., 1159 Park av
Amende, C. G., 319 W. 45th st
Ames, Samuel Patten, 117 W. 71st st
Anderson, H. A. C., 118 E. 86th st
Anderson, James H., Cambridge, Mass.
Anderson, Joseph, 74 W. 92d st
Andrews, Joseph Alfred, 107 Madison av
Anthes, Frederick C., 125 E. 83d st
Armstrong, Robert J., 1804 Lexington av
Armstrong, S. T., 141 Broadway
Armstrong, William, 63 E. 55th st
Aronson, Moses, 101 7th st
Aspell, Edward J., 357 W. 56th st
Aspell, John, 105 Madison av
Assenheimer, Augustus, 323 E. 51st st
Atwood, Charles Edwin, 10 W. 16th st
Auersperg, Prince Francis, 226 W. 78th st
Austin, David P., 12 W. 95th st
Auzal, Ernest W., "The Strathmore," 52d and Broadway
Ayers, Edward A., 127 W. 58th st
Ayres, Winfield, 112 W. 94th st
Bache, James H., 269 W. 52d st
Bachmann, Eugene O., 312 W. 34th st
Bacon, Gorham, 47 W. 54th st
Bailey, Pearce, 52 W. 53d st
Bailey, Theodosus, 77th st and Broadway
Bainbridge, William Seaman, 34 Gramercy Park
Baird, Addison W., 239 W. 70th st
Baker, Henry A., 141 Broadway
Baker, Sara Josephine, 8 W. 91st st
Baldwin, Helen, 53 E. 20th st
Ball, A. Brayton, 42 W. 36th st
Ball, George Martin, 365 W. 28th st
Ballery, George H., 76 W. 47th st
Ballin, Milton J., 57 E. 90th st
Balser, William, 218 E. 13th st
Bandler, Samuel W., 127 E. 61st st
Baner, William L., 72 W. 45th st
Bang, Richard T., 139 W. 11th st
Bangs, L. Bolton, 39 E. 72d st
Banta, Francis M., 304 W. 12th st
Baran, Julius, 134 E. 79th st
Barclay, Hjalmar V., 150 E. 71st st
Bardes, A. C., 317 E. 13th st
Barnes, Justin L., 16 E. 43d st
Barrows, C. C., 8 W. 36th st
Barry, Daniel E., 447 Lexington av
Bartholomew, Henry S., 102 W. 57th st
Bartlett, Frank H., 419 W. 147th st
Bartlett, William A., 7 W. 49th st
Barton, Joshua L., 57 E. 55th st
Baruch, Simon, Hotel Majestic, W. 72d st
Basch, Seymour, 48 E. 63d st
Bastedo, Walter Arthur, 73 W. 49th st
Bates, W. H., 567 Park av
Battle, Thomas Joseph, 460 W. 34th st
Bauer, Frederick M., 225 E. 86th st
Baum, Joseph, 55 E. 93d st
Beach, Ferdinand, 235 W. 75th st
Beck, Alexander W., 336 2d av., bet. 19th and 20th sts
Beck, Carl, 37 E. 31st st
Becker, Philip G., 331 W. 18th st
Beekman, John N., 10 E. 12th st
Bell, George Huston, 54 E. 25th st
Bellamy, Russell, 35 W. 31st st
Benedict, Charles Sumner, 310 W. 102d st
Bensel, Walter, 282 Amsterdam av., near 73d st
Bent, George K., 309 E. 86th st
Berens, T. Passmore, 35 Park av
Berk, Adelbert B., St. Paul building, cor. Ann and Broadway
Berkeley, William N., 40 E. 58th st

Bermingham, Jose Marie De, 106 W. 45th st
Berry, Inslee H., 166 W. 22d st
Bettini-Moise, John, 60 W. 76th st
Beuermann, John A., 1891 Lexington av
Beveridge, J. Wallace, 219 W. 80th st
Bicak, Joseph F., 875 E. 169th st
Bickerton, Thomas Wotton, 656 West End av
Bickham, Warren Stone, 922 Madison av
Bierhoff, Frederic, 53 E. 58th st
Bilhoefer, Andrew J., 79 E. 116th st
Billings, John S., Jr., 32 E. 53d st
Billington, Cornelius E., 85 Madison av
Bingham, James R., 216 W. 103rd st
Bird, Arthur, 159 W. 48th st
Bird, J. T. Joseph, 236 W. 123d st
Birkins, Ernest R., 84 W. 120th st
Bishop, James, 63 E. 56th st
Bishop, Joseph N., 12 W. 38th st
Bishop, Louis Faugeres, 54 W. 55th st
Bissell, Joseph B., 46 W. 55th st
Bissell, Mary Taylor, 21 W. 44th st
Black, Carrie L., 114 W. 123d st
Blake, Joseph A., 601 Madison av
Blank, Marcus I., 1525 Madison av
Blasucci, Ernesto S. L., 127 E. 47th st
Blauvelt, Alonzo, 338 W. 12th st
Bleil, Frederick, 1350 Lexington av
Bleiman, Asher, 206 E. 48th st
Bleything, George D., 27 E. 81st st
Bloch, Moritz, 337 E. 58th st
Blodgett, Frank Jefferson, 140 W. 72d st
Bloom, Selina, 542 Fifth av
Boeker, Hermann H. F., 227 E. 57th st
Bogart, Belmont de Forest, 315 W. 79th st
Bogart, George W., 139 E. 47th st
Bogue, Edward A., 63 W. 48th st
Boldt, Hermann J., 39 E. 61st st
Bond, Hunter A., Ass't Res. Phys. Manhattan State Hosp., Ward's Island
Bonner, Adolph, 2 W. 116th st
Booth, J. Arthur, 46 W. 49th st
Born, R. O., 23 W. 35th st
Bosworth, Francke H., 41 Park av
Bovaird, David, 126 W. 58th st
Bowden, James W., 309 Broadway
Bowles, Fred J., 146 W. 93d st
Boyd, William A., 362 Willis av
Boyer, Arthur A., 12 W. 40th st
Boyer, Arthur I., 356 W. 145th st
Boynton, Perry Sanborn, 103 W. 84th st
Bozeman, Nathan Gross, 167 E. 71st
Bradley, C. Cole, 51 W. 50th st
Bradley, Simeon C., 2700 Marion av
Bradley, William Little, 119 W. 75th st
Bradshaw, Laban L., 55 E. 72d st
Brainin, Simon Michael, 44 E. 112th st
Brandegee, William Partridge, 17 W. 46th st
Brannan, John Winters, 11 W. 12th st
Branth, John Herman, 183 W. 87th st
Breakell, James A., 249 W. 52d st
Breiter, Nathan, 70 Lenox av
Bremner, Samuel K., 163 W. 73d st
Brettauer, Joseph, 45 E. 64th st
Brewer, Charles, 130 W. 66th st
Brickelmaier, Wm. J., 25 W. 36th st
Brickner, Samuel M., 136 W. 85th st
Brickner, Walter M., 30 W. 92d st
Bridges, Arlanden C., 344 W. 28th st
Briganti, P., 214 E. 17th st
Brill, Nathan E., 48 W. 76th st
Brinkmann, Morris W., 54 W. 90th st
Bristol, E. L. Macomb, 46 W. 35th st
Brodhead, George Livingston, 110 W. 57th st
Bronson, Edward Bennet, 10 W. 49th st
Brooks, G. Frederick, 247 Central Park, West
Brooks, Harlow, 44 W. 9th st
Brooks, Henry T., N. Y. Post-Graduate Hosp., 20th st and 2d av
Bross, William R., 120 Broadway
Brothers, Abram, 112 E. 61st st
Broughton, William R., 345 Madison av
Broun, Leroy, 70 W. 82d
Brouner, Walter Brooks, 256 W. 12th st
Browd, Ephraim K., 275 E. Broadway
Brown, Augustus H., 262 W. 136th st
Brown, Ethel Doty, 128 Lexington av
Brown, F. Tilden, 14 E. 58th st
Brown, J. Morris, Col.-Ass't. Surg.-Gen. U. S. A., 391 Washington st
Brown, Lewis Henry, 573 E 156th st
Brown, Mary Hess, 528 W. 123d st
Brown, Richard Ewell, 111 W. 55th st
Brown, Samuel Alburtus, 23 E. 44th st
Brown, William Bedford, 214 W. 44th st
Bruce, Charles E., 2 E. 127th st
Brugman, Albert F., 201 W. 139th st cor. 7th av
Brush, Edward F., Mt. Vernon

Bryan, J. Conger, 51 W. 91st st
Bryant, Joseph D., 32 W. 48th st
Büchler, Augustus F., 124 W. 58th st
Buck, Albert H., 48 W. 40th st
Buck, Francis D., 158 W. 48th st
Bucklin, Charles A., 237 W. 45th st
Buffum, Thomas Bellows, 128 W. 66th st
Bulkley, L Duncan, 531 Madison av
Bull, Charles Stedman, 47 W. 36th st
Bull, William T., 35 W. 35th st
Bullard, W. Duff, 302 Madison av
Bullard, William E., Larchmont-on-the-Sound
Bullinger, J. A., 120 E. 57th st
Burch, T. Hamilton, 106 W 57th st
Burgheim, L,, 176 E. 79th st
Burgtorf, Theodore R., 93 2d av
Burke, Michael Joseph, 164 Hester st
Burkelman, Arnold, 3 Charlton st
Burnham, Herbert D., 313 Lenox av
Burrall, Frederick A., 48 W. 17th st
Burstein, Maurice J., 179 Henry st
Burt, Stephen Smith, 207 W. 44th st
Burtenshaw, J. H., 381 West End av., cor. 78th st
Busche, Thomas W,, 241 E. 18th st
Bush, Pine E., 45 Charlton st
Bushong, Chas. H., 59 W. 92d st
Butler, George H., 964 5th av
Butts, H. Hoyle, 313 Madison av
Byrne, Joseph Henry, 345 W. 51st st
Cabot, Follen, 129 E. 31st st
Cabot, John, 1416 Adams st., Hoboken, N. J.
Cady, Herbert W. F., 25 W. 36th st
Caille, Augustus, 753 Madison av., near 65th st
Calhoun, William Caldwell, 205 W 107th st
Callan, Peter A., 35 W. 38th st
Cammann, D. M., University Club
Campbell, George Busby, Manhattan State Hosp., Ward's Island
Carling, John, 126 E. 34th st
Carlisle, Chester Lee, Manhattan State Hospital, Ward's Island
Carman, Albro R., 27 W. 127th st
Carpenter, Alfred C., 219 E. 19th st
Carpenter, Elon N., 110 W. 57th st
Carpenter, Frank B., The Allston, 17 E. 38th st
Carr, David C., 2 E. 127th st
Carr, Walter Lester, 68 W. 51st st
Carroll, C. C., 22 W. 32d st
Carter, De Lancey, 1030 Park av
Carter, H. Skelton, 130 E. 24th st
Carter, Henry Walton, 148 E. 35th st
Carter, Herbert Swift, 66 W. 55th st
Carter, William Wesley, 69 W. 50th st
Carver, J. Henry, 37 W. 39th st
Cassell, James Wilson, 152 W. 132d st
Caswell, Wm. H., 150 W. 58th st
Catherwood, Clinton H., 241 W. 75th st
Cauldwell, Charles M., 16 W. 54th st
Cereseto, Giovanni P., 99 McDougall st
Chadbourne, Edwin R., 745 S. Orange Grove av., Pasadena, Cal.
Chalmers, Thomas C., 5 W. 8th st
Chamberlin, Edwin Crosby, 5 W. 82d st
Chamberlin, Frank Warren, 139 Convent av
Chambers, P. Flewellen, 49 W. 57th st
Chandler, George F., Kingston, N. Y.
Chapin, Amory, N. Y. Athletic Club
Chapin, Henry Dwight, 51 W. 51st st
Chapin, Warren B., 252 W. 103d st
Chappell, Walter Franklin, 7 E. 55th st
Chase, George T., 238 W. 127th st
Chase, William Earle, 139 E. 47th st
Chauveau, Jean F., 31 W. 60th st
Cheesman, Timothy Matlack, Garrison-on-Hudson, N. Y.
Chittenden, Arthur Smith, 166 W. 74th st
Chmelicek-Luhan, Joseph F., 220 E. 72d st
Chrystie, T. M. Ludlow, 204 W. 55th st
Churchill, Frank, 317 E. 86th st
Churchill, S. Wells, 165 W. 64th st
Cilley, Arthur H., 152 E. 34th st
Cisin, Morris, 121 Rivington st
Claiborne, John Herbert, 39 W. 36th st
Clark, J. Bayard, 46 W. 48th st
Clark, Pierce, 62 W. 58th st
Clark, William Brewster, 50 E. 31st st
Clarke, Walter J., 2297 7th av
Clarke, William Cogswell, 200 W. 57th st
Cleaveland, Trumbull W., 45 W. 50th st
Cleaves, Margaret A., 79 Madison av., cor. 28th st
Clemens, James B., 19 E. 38th st
Clements, Geo. Place, 60 St. Nicholas av
Cleveland, Clement, 59 W. 38th st
Close, Seth D., 636 E. 143d st
Coakley, Cornelius Godfrey, 49 W. 58th st
Coe, Henry C., 8 W. 76th st

Cobb, George Henry, South Orange, N. J.
Coffin, Lewis A., 49 W. 50th st
Coggeshall, Henry, 40 E. 58th st
Cohen, Martin, 205 E. 115th st
Cohn, Felix, 717 Madison av
Cohn, Louis, 1309 Madison av
Cohn, Salo, 1857 Madison av
Cole, Carter S., 122 W. 73d st
Cole, Lewis Gregory, 936 W. End av
Cole, Palmer C., 247 W. 42d st
Coleman, Warren, 58 W. 55th st
Coley, Wm. B., 5 Park av
Colles, Christopher J., 15 E. 29th st
Collings, Howard P., Hot Springs, Ark.
Collins, Chas. Farnham, 50 W. 55th st
Collins, Joseph, 32 W. 38th st
Collyer, Herman L., 109 E. 54th st
Coman, John S., 860 E. 165th st
Combes, Frank C., 165 E. 116th st
Comegys, Howard Ogle, 170 E. 96th st
Cone, Henry A., 292 Central Park, West
Conner, Lewis A., 53 E. 49th st
Connor, Henry Rock, 256 W. 34th st
Connors, John F., 117 W. 83d st
Conover, William S., 240 W. 132d st
Constable, Herbert L., 72 W. 48th st
Conterno, George W., 137 McDougal st
Cook, Finley R., 96 Park av
Cook, Stephen Guernsey, 111 W. 12th st
Corning, J. Leonard, 53 W. 38th st
Cosby, John B., 351 W. 51st st
Costigan, Gregory, 351 W 32d st
Cotton, Mary H., 320 Manhattan av
Coughlin, John H., 307 E. Broadway
Cowen, William, 35 E. 60th st
Cox, Rowland, Jr., 547 Madison av
Cragin, Edwin B., 10 W. 50th st
Crandall, Floyd M., 113 W. 95th st
Crary, George Waldo, 771 Madison av
Creevey, George Mason, 515 Madison av., cor. 53d st
Cremin, Patrick W., 856 Lexington av
Crispin, Antonio Maria, 214 E. 69th st
Crofoot, Wellington A., Manhattan state Hospital
Cronin, John J., 433 W. 51st st
Crook, James King, 783 Madison av
Crowley, Michael F. J., 304 7th av
Culbert, William Ledlie, 54 E. 34th st
Culler, Frederick W., 35 W. 60th st
Culver, Everett Mallory, 36 W. 35th st
Cunningham, Richard H., 200 W. 56th st
Curran, Martin William, 154 E. 72d st
Curran, Stanley Douglas, 65 W. 99th st
Curtis, B. Farquhar, 7 E. 41st st
Curtis, Charles T., 160 W. 106th st
Curtis, Edward, 33 W. 69th st., 120 Broadway
Curtis, H. Holbrook, 118 Madison av
Curtis, John G., 327 W. 58th st
Cushman, William F., 172 Ninth av
Cutler, Colman Ward, 52 W. 53d st
Cutler, Condict W., 135 W. 76th st
Dadirrian, Markar G., 73 Lexington av., bet. 25th and 26th sts
Daley, Robert M., 43 E. 27th st
Dallas, Alexander, Bayonne, N. J.
Dalton, W. R. I., 813 N. 5th st., Tacoma, Wash.
Dana, Charles L., 53 W. 53d st
Dana, Samuel Wood, 162 W. 94th st
Daniel, Annie S., 327 E. 15th st
Daniels, Frank Herbert, 103 W. 122d st
Danne, Frederic, 44 W. 50th st
Darlington, Thomas, Kingsbridge
Davidson, Joseph, 173 E. 82d st
Davin, John P., 117 W. 76th st
Davis, Albert A., 119 E. 62d st
Davis, Asa B., 42 E. 35th st
Davis, A. Edward, 42 E. 41st st
Davis, E. Webster, 153 W. 44th st
Davis, Fellowes, Jr., 58 W. 47th st
Davis, Francis William, 211 W. 12th st
Davis, Willis Oscar, 2405 Seventh av
Dawbarn, Robert H. M., 105 W. 74th st
Dayton, Hughes, 126 W. 48th st
Deane, William C., 114 E. 60th st
De Garmo, William B., 56 W. 36th st
Delacroix, Arthur Charles, Manhattan State Hospital, Ward's Island
Delafield, Francis, 12 W. 32d st
Delavan, D. Bryson, 1 E. 33d st
Delphey, Eden V., 400 W. 57th st
Deming, William Champion, Westchester, N. Y. City
Dench, Edward B., 17 W. 46th st
Denchfield, L. J., 1 W. 115th st
Denhard, Charles E., 90 Madison st
Denig, Rudolf, 30 W. 59th st
Dennett, William Sawyer, 8 E. 49th st
Dennis, Frederic S., 542 Madison av
Dent, Emmet C., Manhattan State Hosp., West, Ward's Island
Denton, Myron Preston, 33 E. 33d st
De Plasse, Louis, 27 and 29 E. 27th st

Derby, Richard H., 9 W. 35th st
Dessar, Leonard A., 154 W. 58th st
Dessau, S. Henry, 144 W. 85th st
Dew, J. Harvie, 65 W. 68th st
Dexter, John Milton, 109 W. 87th st
Deynard, A. Britton, 333 W. 51st st
Dieffenbach, R. G. L., 224 E. 48th st
Dillingham, Frederick H., 76 W. 85th st
Dillon, Joseph A., 967 Lexington av
Dixon, George A., 15 W. 49th st
Dixon, George Sloan, 124 Lexington av
Dockstader, Charles H., 483 Manhattan av
D'oench, Frederick E., 49 E. 58th st
Dold, Wm. Elliott, 1125 Madison av
Dorman, Franklin A., 57 W. 58th st
Dorn, John H., 39½ Washington Sq., South
Dorning, John, 111 W. 48th st
Doty, George E., 216 W. 38th st
Doubleday, Jas. Stewart, Amityville, Long Island
Douglas, John, 145 W. 81st st
Douglas, O. B., Concord, N. H.
Douglass, H. Beaman, 40 E. 41st st
Dounce, Jay William, 8 W. 92d st
Dow, Edmund Le Roy, 49 W. 57th st
Dowd, Charles N., 135 W. 73d st
Downes, William A., 47 W. 44th st
Downey, Martin, 119 E. 26th st
Drake, William F., 300 Central Pk., West
Draper, William K., 121 E. 36th st
Drayton, Henry S., 70 Fifth av
Duane, Alexander, 49 E. 30th st
Dudley, A. Palmer, 678 Madison av
Duel, Arthur Baldwin, 254 Madison av
Dunckel, Walter Adams, 150 W. 44th st
Dunham, Edward K., 338 E. 26th st
Dunham, Theodore, 305 W. 76th st
Dunn, Thomas Joseph, 2735 Webster av
Dunseith, John Franklin, 354 W. 42d st
Durant, Ghislani. In Europe
Durham, Albert, 10 W. 16th st
Dwight, Jonathan, Jr., 2 E. 34th st
Dwyer, John, 182 E. 111th st
Eastman, Robert Watts, 140 W. 76th st
Eaton, Louise, 462 W. 145th st
Echeverria, Martin John, 141 W. 73d st
Edebohls, George M., 59 W. 49th st
Edgar J. Clifton, 50 E. 34th st
Edgerton, James Ives, 36 W. 35th st
Edlich, Theodore J., 155 E. 45th st
Egan, William H., 371 W. 32d st
Ehrhart, Paul, 351 W. 29th st
Eichler, Philip, 1810 Lexington av
Einhorn, Max, 20 E. 63d st
Eisnig, Eugene H., 20 E. 60th st
Eliot, Ellsworth, 48 W. 36th st
Elliot, George T., 36 E. 35th st
Elliott, George R., 48 E. 26th st
Ellison, C. Ruxton, 206 E. 116th st
Ellison, Saram R., 118 W. 103d st
Elsberg, Charles Albert, 105 E. 57th st
Elwert, Charles P., 116 W. 47th st
Ely, Albert H., 47 W. 56th st
Ely, Leonard W., 451 West End av
Emans, J. Seymour, 78 Irving pl., cor. 19th st
Emerson, J. Baxter, 20 E. 30th st
Emerson, J. H., 81 Madison av
Emmert, Frank N., 110 W. 132d st
Emmet, Bache, 18 E. 30th st
Emmet, John Duncan, 91 Madison av
Emmet, Thos. Addis, 91 Madison av
Ennist, Floyd Bonesteel, 248 W. 105th st
Eno, Henry C., Saugatuck, Ct.
Erdmann, John F., 60 W. 52d st
Erlwein, Michael, 1349 Columbus av
Ernst, Peter H., 141 E. 16th st
Ettinger, Henry, 2150 Fifth av
Evans, Evan Morton, 38 W. 48th st
Evans, G. A., 337 W. 15th st
Evans, Samuel M., 115 E. 39th st
Evarts, Herman Crocker, Manhattan State Hosp., West, Ward's Island
Eversfield, Frank J., 226 W. 21st st
Ewing, James, 260 W. 57th st
Ewing, W. A., 134 W. 58th st
Fairbank, C. O., Petrolea, Ontario
Farr, Edgar Horace, 321 W. 46th st
Farrar, J. N., 1271 Broadway
Farrington, J. O., 1991 Madison av
Farrington, William H., Astor House
Feinberg, Israel L., 1716 Madison av
Feinberg, S., 148 E. 81st st
Fendler, Amelia M., 317 E. 89th st
Fenn, Elisha, 357 W. 116th st
Ferguson, Frank, 20 W. 38th st
Ferguson, James Alexander, Lind av., Highbridge
Ferguson, J. Bruce, 123 E. 19th st
Ferguson, James Francis, 168 Lexington av
Ferguson, Jeremiah Sweetser, 330 W. 28th st
Ferrer, Jose M, 441 Park av
Ferris, Albert Warren, 343 Madison av
Fielder, Frank S., 2 W. 82d st
Fielding, Roberta Lee, 20 E. 78th st
Fields, William J., 250 W. 88th st

Finch, Edward B., 9 W. 24th st
Fischer, Charles Sumner, 144 E. 38th st
Fischer, Charles S., Jr, 201 W. 118th st
Fischer, Louis, 65 E. 90th st
Fischer, Siegfried, 314 E. 15th st
Fischlowitz, Gustav Grant, 1298 Madison av
Fisher, Edward D., 19 W. 52d st
Fishmann, Arthur, 118 Rivington st
Fisk, Arthur Lyman, 41 W. 50th st
Fiske, James Porter, 1690 Broadway
Fitch, Allen, 38 W. 56th st
Fitch, Charles W., 640 Madison av
Fitzgerald, Fred J. C., 127 E. 24th st
Fitzhugh, Patrick Henry, 36 W. 35th st
Fleming, Walter M., Hotel Victoria
Flesch, Berthold, 307 E. 87th st
Floersheim, Samuel, 210 E. 46th st
Floy, Frederic H., 381 West End av
Fluhrer, Willam F., 479 Fifth av., cor. 41st st
Foerster, Francis, 926 Madison av
Foland, John P., 511 Hudson st
Foote, Edward Milton, 67 W. 48th st
Forbes, Henry Hall, 96 Park av
Ford, Charles M., 523 W. 141st st
Ford, James C., 233 E. 104th st
Fordyce, John A., 66 Park av
Foster, Matthias Lanckton, 542 Fifth av., cor. 45th st
Fowler, Edmund Prince, 391 West End av., near 79th st
Fowler, Edward Payson, 38 W. 40th st
Fowler, George B., 18 E. 58th st
Fowler, Samuel W., 116 W. 103d st
Fox, Andrew Jackson, 73 E. 54th st
Fox, George Henry, 18 E. 31st st
Fraenkel, Joseph, 46 E. 75th st
Francke, P. F., 104 E. 60th st
Frank, Isaiah, 130 E. 64th st
Frank, John, 221 E. 86th st
Frankel, Edward, 217 E. 17th st
Frankenberg, J. H., 56 E. 73d st
Frauenthal, Herman C., 146 W. 72d st
Freeborn, George C., 215 W. 70th st
Freedman, Joseph, 26 E. 122d st
Freeman, Alpheus, 123 E. 74th st
Freeman, Rowland G., 205 W. 57th st
Freiberger, Leopold, 326 E. 65th st
Freid, Moosha B., 668 E. 142d st
Fridenberg, Edward, 242 Lenox av., near 122d st
Fridenberg, Percy H, 114 W. 126th st
Friedman, Louis, 42 W. 115th st
Friedman, Maurice, 103 E. 29th st
Frischbier, Charles Paul, Manhattan State Hosp., West, Ward's Island
Frissell, Lewis Fox, 113 E. 56th st
Froehlich, David, 117 E. 80th st
Fuchs, Frederick Louis, 10 St. Mark's pl
Fuld, Joseph E., 169 E. 82d st
Fuller, Eugene, 252 Lexington av
Fuller, Robert M., 136 W. 42d st
Furness, W. J., 271 W. 134th st
Galbraith, Anna M., 15 W. 91st st
Gallant, A. Ernest, 60 W. 56th st
Gant, Samuel G., 43 W. 52d st
Gardner, Alfred W., 325 W. 89th st
Gardner, Arthur H., 939 Madison av
Gardner, William C., 121 W. 71st st
Gardner, Wm. S., 101 W. 84th st
Garrigue, Evelyn, 172 W. 79th st
Garrigues, Henry J., 184 E. 72d st
Gazzam, Edwin Van Deusen, 233 W. 83d st
Geoghan, William, 78 W. 82d st
Gerster, Arpad G., 34 E. 75th st
Gibb, W. Travis, 55 W. 38th st
Gibney, Homer, 31 W. 36th st
Gibney, Virgil P., 16 Park av
Gibson, Chas. Langdon, 48 W. 49th st
Gibson, Lewis M., 157 W. 81st st
Gignoux, Harriet A., 72 W. 45th st
Gilday, Walter C., 32 W. 36th st
Giles, J. Edward, 107 E. 23d st
Gilfillan, W. Whitehead, 24 W. 59th st
Gillette, Walter R., 24 W. 40th st
Ginnasi, Francis, 118 Macdougal st
Gleitsmann, Joseph Wm., 46 E. 25th st
Gluck, Samuel, 1041 Madison av
Gnichtel, A. Lawrence, 257 W. 44th st
Godson, George H., 301 W. 114th st
Goetchius, Harry D. 147 E. 51st st
Goldan, S. Ormond, 18 W. 70th st
Goldenberg, Herman, "The Leonori." 63d st and Mad. av
Goldlust, James, 153 E. 90th st
Goldmark, Carl, 144 W. 127th st
Goldwater, Abraham L., 66 E. 112th st
Gonzales, Thomas Arthur, 231 E. 94th st
Goodall, William Ainslie, 565 E. 158th st
Goode, Ernest W., 367 W. 48th st
Goodman, Abraham Lincoln, 263 W. 113th st
Goodman, Charles, 243 E. B'way
Goodman, Henry L., 1143 Lexington av
Goodwillie, D. H., 5 W. 31st st
Gordon, Bernard, 152 Henry

Gorman, John D., 326 E. 67th st
Gorton, Orren A., Sherburne, N. Y.
Gottheil, William S., 144 W. 48th st
Gottlieb, J. Adelphi, 30 W. 119th st
Gould, David Bartlett, 225 W. 15th st
Graber, Sidney S., 185 E. 71st st
Grandin, Egbert H., 116 W. 76th st
Grant, Gabriel, 22 E. 49th st
Gray, Rollin B., 202 W. 81st st
Greanelle, William J., Loring pl., University Heights
Greeff, J. G. William, 24 W. 91st st
Greene, Robert Holmes, 47 W. 38th st
Griffin, E. Harrison, 112 W. 45th st
Griffin, Henry Arthur, 346 Broadway
Griswold, Henry, 70 W. 48th st
Gross, Morris, 315 Second av
Grosvenor, Frank L., Manhattan State Hosp., Ward's Island
Gruening, Emil, 36 E. 57th st
Guck, Arago J., 401 Hancock st., Brooklyn
Guiteras, Ramon, 75 W. 55th st
Gunning, Josephus Henry, 640 Madison av
Guntzer, J. Henry, 345 E. 86th st
Guth, William C., 253 E. 71st st
Guttman, Frederick, 228 W. 132d st
Guttman, John, 333 E. 4th st
Gwathmey, James Taylor, 124 E. 16th st cor. Irving pl
Gwyer, Fred W., 130 E. 38th st
Habersack, Charles Alex., 840 Trinity av
Hadden, Alexander, 155 E. 51st st
Hale, Henry Ewing, Jr., 150 W. 59th st
Hall, Robert Wellington, 118 E. 29th
Hall, William H., 129 E. 54th st
Hallock, Luther Reeve, 217 W. 127th st
Hallock, Silas F., 36 E. 65th st
Halsey, Frank Spencer, 108 W. 74th st
Halsey, Robert Hurtin, 57 W. 58th st
Hamburg, D. J., 157 W. 118th st
Hamilton, Allan McLane, 44 E. 29th st
Hamlen, George Dempster, 8 W. 36th st
Hammond, Graeme M., 60 W. 55th st
Hance, Irwin H., Lakewood, N. J.
Hardy, Sara Browne Belcher, 419 W. 118th st
Hare, George Ray, 107 E. 23d st
Harlow, Ellwood, 13 E. 77th st
Harris, Thomas J., 117 E. 40th st
Harrison, Calvin L., 346 Broadway
Harrison, George Tucker, 63 W. 51st st
Hartwell, John A., 50 E. 53d st
Haskin, William Henry, 42 E. 41st st
Haupt, Louis, 232 E. 19th st
Haviland, Clarence Floyd, Manhattan State Hosp., Ward's Island
Hawes, Walker A., 787 Lexington av
Hawkes, Forbes, 42 E. 26th st
Hayden, James R., 107 W. 55th st
Hayes, William Van Valzah, 10 E. 43d st
Haynes, Irving S., 1125 Madison av
Hazen, Allen, 174 W. 87th st
Hazen, Henry Comstock, 66 W. 56th st
Healy, Joseph S., 2886 Bailey av., Kingsbridge
Hedden, Jesse W., 149 E. 21st st
Heiman, Henry, 56 W. 120th st
Heitzmann, Louis, 110 W. 78th st
Held, Ruben Johnson, 320 Central Park, West, cor. 92d st
Hellenstein, Herman, 275 7th st
Henna, J. Julio, 8 W. 40th st
Henry, John P., 329 W. 58th st
Henry, Nelson H., 48 W. 11th st
Henson, Henry B., 320 W. 125th st
Herbert, Henry, Bedford Station, N. Y.
Herman, Henry, 937 Madison av
Herrick, Everett, 126 Madison av
Herold, Justin, 325 E. 87th st
Herrick, William Post, 30 W. 54th st
Herter, Christian A., 819 Madison av
Herzfeld, Alfred A., 224 W. 24th st
Herzig, Arthur J., 204 W. 119th st
Heuel, Emil, 702 E. 138th st
Heuel, Frank, 117 W. 79th st
Heywood, George, 62 Madison av
Hibbard, William E., Pasadena, Cal.
Hibbs, Russell A., 136 Madison av
Hickey, Charles M., 155 E. 34th st
Hicks, Elias Peter, 940 Flatbush av., Brooklyn
Higgins, Joseph J., 141 W. 76th st
Hill Edmund Yard, 111 W. 71st st
Hill, Isadore Leon, 774 Madison av
Hillis, Thomas J., 51 Charlton st
Hills, Alfred Kimball, 669 Fifth av
Hills, Arthur T., Hotel Endicott, 101 W. 81st st
Himowich, Adolph A., 130 Henry st
Hirons, Gardner, 131 W. 136th st
Hirsch, William, 52 E. 64th st
Hitchcock, Charles, 57 W. 36th st
Hitchcock, Edwin F., 301 W. 139th st
Hitchcock, Urban G., 51 W. 29th st
Hoag, Ward B., 146 W. 87th st
Hobbs, Angier Bailey, 512 W. 151st st

Hoch, John, 162 E. 64th st
Hochheimer, Emanuel, 1311 Madison av
Hoeber, Emil W., 612 Lexington av
Hoffmann, Charles, 1219 Madison av
Hoffmann, Julius L. P., 152 E. 81st st
Holden, T. N., 294 E. Broadway
Holden, Ward A., 43 W. 48th st
Holder, Oscar Howe, 66 Park av
Holgate, Thomas H., 206 W. 14th st
Hollis, Austin W., 111 W. 47th st
Hollister, Frank Canfield, 264 W. 77th st
Holmes, Edwin, Englewood, N. J.
Holmes, Geo. W., 222 W. 70th st
Holmes, John F., 1043 Boston Road
Holmes, Martha C., Waterbury, Ct.
Holt, L. Emmett, 14 W. 55th st
Honegger, Oscar P., 171 E. 71st st
Hoover, Franklin P., 832 Riverside av., Jacksonville, Fla.
Hope, George B., 133 W. 72d st
Hopkins, Frank Tucker, 200 W. 113th st
Hopkins, J. Swinburne, 52 W. 84th st
Horn, John, 255 E. Broadway
Hotchkiss, Lucius W., 37 W. 48th st
Houghton, H. Seymour, 301 W. 88th st
Howell, Hampton P., 65 W. 70th st
Hubbard, Ernest V., 138 W. 74th st
Hubbard, William Norris, 53 W. 54th st
Hubbell, Marvin D., 245 W. 135th st
Hubby, Lester Mead, 40 W. 84th st
Huber, Francis, 209 E. 17th st
Huber, John Bessner, 44 E. 64th st
Huber, Joseph, 113 E. Broadway
Huddleston, John H., 126 W. 85th st
Hudson, Walter G., 73 W. 131st st
Huhner, Max, 64 E. 58th st
Hull, Joseph J., "The Albany," Broadway and 51st st
Hume, Frederick T., 107 W. 75th st
Hume, William A., 107 W. 75th st
Hunt, Edward Livingston, 62 W. 50th st
Hunt, James Ramsey, 102 E. 57th st
Hunter, Dwight W., 80 W. 40th st
Hunter, George T., 332 W. 33d st
Huntington, Abel, 346 Broadway
Hurd, Edward F., Anthony av and 176th st
Hurd, Lee Maidment, 19 E. 38th st
Huson, Martha B., 237 E. 86th st
Hustace, Francis, 413 Madison av
Hutchinson, Anna Earl, Manhattan State Hosp., West, Ward's Island
Hyde, Frederick E., 20 W. 53d st
Hymanson, A., 232 Henry st
Illoway, Henry, 1113 Madison av
Inglehart, Asa S., 77 W. 55th st
Irwin, Frank Newton, 10 W. 39th st
Irwin, John Arthur, 14 W. 29th st
Irwin, S. Nelson, 217 W. 42d st
Isaacs, Archibald E., 240 E. Broadway
Ives, Frederick M., 111 E. 19th st
Jackson, Charles Ross, 220 W. 107th st
Jackson, Charles Warren, 130 W. 82d st
Jackson, Frank W., 68 W. 55th st
Jackson, George Thomas, 15 E. 29th st
Jackson, John Alexander, 47 W. 44th st
Jackson, Meyer, 180 E. 79th st
Jackson, Moses Jose, 125 E. 84th st
Jackson, Victor Hugo, 240 Lenox av
Jacob, Isidore H., 1228 Madison av
Jacobi, Abraham, 19 E. 47th st
Jacobi, Mary Putnam, 19 E. 47th st
Jacobsohn, William, 120 W. 98th st
Jacobson, Sidney Dattner, "The Mestaigne," 682 E. 138th st
Jacobus, Arthur Middleton, "The Rutland," 260 W. 57th st
Jacoby, George W., 605 Madison av
Jacoby, J. Ralph, 605 Madison av
Jagger, A. W., 410 Amity, Flushing
Jahn, Otto August, 218 E. 45th st
James, Robert C., 58 W. 55th st
James, Walter B., 17 W. 54th st
Janeway, Edward G., 36 W. 40th st
Janvrin, Joseph E., 191 Madison av
Jarecky, Herman, 115 W. 121st st
Jarman, George Wallace, 54 W. 76th st
Jay, John C., Century Club
Jeffrey, Alexander McL., 133 E. 39th st
Jelliffe, Smith Ely, 231 W. 71st st
Jenkins, William T., 24 State st
Jennings, D. D., 226 E. 17th st
Jennings, Walter Barry, 113 E. 28th st
Jewett, Charles Taylor, 162 W. 22d st
Jewett, Mary B., 30 E. 57th st
Johnson, Alexander B., 12 E. 58th st
Johnson, Henrietta Pauline, 206 E. 15th st
Johnson, John D., 130 W. 104th st
Johnson, Philip Van K., 126 W. 48th st
Johnson, W. H., 103 W. 29th st
Johnston, James C., 64 E. 56th st
Jones, Catesby, 18 W. 35th st
Jones, Charles N. Dixon, 62 E. 86th st

Jones, Israel C., Med. Supt. Home for Incurables, Fordham
Jones, Lucy C., 212 E. 53d st
Jones S. Beach, 12 E. 33d st
Jordan, C. H. Richard, 47 W. 44th st
Judd, Aspinwall, 295 Central Park, West
Judson, Adoniram B., Room 528, 1 Madison av
Kakels, Moses S., 71 E. 66th st
Kalish, Richard, 36 W. 47th st
Kammerer, Fred, 51 E. 66th st
Kane, Arthur M., 349 W. 34th st
Kantor, William L., 746 E. 142d st
Kantrovitz, Adolph, 1785 Madison av
Karibe, Ruel B., 246 W. 137th st
Katzenbach, W. H., 17 W. 45th st
Katzenberg, Meyer, 1315 Madison av
Katzenmayer, Guido, 625 Lexington av
Kaufman, J., 52 E. 58th st
Keays, Frederick Love, 200 W. 57th st
Keller, Frederick C., 411 W. 43d st
Kellogg, Alexander A., 158 W. 53d st
Kelogg, Edward L., 104 W. 58th st
Kellogg, Theodore H., cor. Riverdale Lane and Albany Post road, Riverdale, N. Y. City
Kelly, Thomas, 357 W. 57th st
Kelsey, Charles B., 18 E. 29th st
Kemp, Robert Coleman, 107 E. 57th st
Kempner, Soloman Horace, 168 W. 130th st
Kenefick, Joseph Aloysius, 29 W. 36th st
Kennedy, John M., 250 W. 99th st
Kennedy, John T., 107 E. 29th st
Kenyon, James H., 47 W. 44th st
Kenyon, Thomas A., 312 Manhattan av., N. E. cor. 113th st
Kerley, Charles Gilmore, 113 W. 83d st
Kershner, Edward, 1 E. 39th st
Kessler, Adolph, 906 Park av
Keune, Theodore, 70 W. 120th st
Keyes, Edward L., 109 E. 34th st
Khodoff, Jacob S., 1421 Madison av
Kilham, Eleanor B., 121 E. 35th st
Kiliani, Otto G. T., 116 E. 57th st
Kilmer, Theron Wendell, 165 W. 85th st
Kilroe, Edward P., 472 West End av
Kimball, Reuel B., 15 E. 41st st
Kinch, Charles A., 273 W. 70th st
Kindred, J. Joseph, 1125 Madison av
King, David F., 651 Lexington av
King, Thomas Armstrong, 139 Lexington av
Kingsbury, Jerome, 349 W. 34th st
Kinney, Charles W., 151 W. 93d st
Kinnicut, Francis P., 39 E. 35th st
Kip, Isaac L., 448 5th av
Klotz, Hermann G., 42 E. 22d st
Klotz, Walter C., 126 W. 45th st
Knapp, Arnold, 26 W. 40th st
Knapp, Herman, 26 W. 40th st
Knapp, John B., 35 W. 75th st
Knapp, John Rudolph, Ward's Island
Knapp, Mark Israel, 136 E. 78th st
Kneer, Ferdinand G., 236 W. 51st st
Knickerbocker, George S., 145 W. 128th st
Knight, Charles H., 147 W. 57th st
Knight, E. Helen, "The Montana," 35 Mt. Morris av. cor. 124th st
Knipe, George, 353 W. 24th st
Knopf, S. A., 16 W. 95th st
Koempel, R. A., 251 E. 86th st., bet. 2d and 3d avs
Koester, Henry F., 1159 Park av
Kohn, Albert, 122 E. 58th st
Kohn, Sophie Kupfer, 132 Manhattan av
Koles, Henry M., 109 E. 116th st
Koller, Carl, 715 Madison av
Kopetzky, Samuel Joseph, 290 W. 137th st
Koplik, Henry, 692 Madison av
Kosmak, Geo. W., 23 E. 93d st
Kraft, Frederic De, 249 W. 54th st
Kremer, Carl F., 112 E. 57th st
Krollpeiffer, Henry, 130 E. 70th st
Krug, Florian, 13 E. 41st st
Kubin, Wilhelm K., 112 W. 120th st
Kudlich, Herman F., 153 W. 21st st
Kunitzer, Robert, 188 Lenox av
Kunz, George W., 174 E. 64th st
Kunz, Hugo, 51 W. 113th st
Kunz, Louis, 48 W. 119th st
Labadie, Ferréol T., 361 W. 27th st
Ladinski, Louis J., 1289 Madison av
La Fétra, Linnaeus Bedford, 58 W. 58th st
Laighton, Florence Marion, 8 W. 91st st
Lambert, Alexander, 125 E. 36th st
Lambert, Charles E., 135 W. 104th st
Lambert, Edward W., 126 E. 39th st
Lambert, Walter Eyre, 8 W. 35th st
Landesmann, M., 309 E. 4th st
Landsman, Samuel M., 220 E. 19th st
Langmann, Gustav, 121 W. 57th st
Langstroth, Francis Ward, Jr., 211 W. 101st st
Lapowski, Boleslaw, 28 W. 59th st
Latham, James R., 203 W. 11th st
Law, Frederick M., 141 W. 122d st
Law, James, 19 E. 127th st

Lawrence, George Alfred, 784 Park av
Leale, Charles A., 604 Madison av
Le Boutillier, William G., 45 W. 50th st
Lederman, E. D., 38 E. 60th st
Liederman, M. D., 38 E. 60th st
Lee, Burton James, 128 E. 73d st
Lee, Charles, 94 Pelham st., Newport, R. I.
Lee, Edward Wallace, 71 Central Park West
Lee, Elmer, 127 W. 58th st
Lee, George Bolling, 215 W. 43d st
Lee, Henry Thomas, 515 Madison av
Le Fevre, Egbert, 52 W. 56th st
Lefferts, George M., 212 Madison av
Lefkovics, Bartholomew, 301 E. 10th st
Lehane, Timothy D., 245 E. 90th st
Lellmann, Carl H., 73 E. 66th st
Lennon, John, 247 E. 86th st
Leo, S. Newton, 103 W. 55th st
Léon, Alexis Marcy, 79 E. 56th st
Leonard, A. D., 27 E. 30th st
Leonard, Z. L., 19 W. 120th st
Lesser, A. Mona, 19 E. 61st st
Leszynsky, William M., 56 E. 58th st
Leverty, Alexander S., 117 E. 114th st
Leviseur, Frederick J., 741 Madison av
Levitt, Marcus Jacob, 135 E. 116th st
Levy, David H., 134 W. 104th st
Lewi, Emily, 35 Mt. Morris av
Lewi, Maurice J., 31 W. 84th st
Lewinthal, David Clarence, 145 2d av
Lewis, Charles Henry, 51 W. 58th st
Lewis, Daniel, 252 Madison av., near 38th st
Lewis, Frank N., 35 W. 36th st
Lewis, Robert, Jr., 48 W. 40th st
Lewis, William Jerauld, 319 W. 116th st
Libman, Emanuel, 180 E. 64th st
Liddle, Herbert James, 68 W. 37th st
Liell, Edward N., Jacksonville, Fla.
Lilienthal, Howard, 766 Madison av
Lines, Ernest H., 346 Broadway
Lippert, Frieda E., 147 Av B
Lively, William M., 216 W. 28th st
Livingston, E. P., 189 Convent av
Lloyd, Samuel, 12 W. 50th st
Lobenstine, Ralph W., 447 W. 59th st
Locke, Anna Willard, 109 W. 82d st
Lockwood, George Roe, 18 E. 52d st
Long, Eli, Jr., 59 W. 97th st
Loomis, H. P., 58 E. 34th st
Lorenze, Edward J., 1658 Lexington av
Lotz, George Carl, 311 E. 10th st
Loughran, Robert L., 824 Lexington av
Love, Isaac N., 537 Fifth av
Lovell, Frederick S., 129 E. 40th st
Luckett, William H., 112 W. 119th st
Ludbrook, W. Wallis, 426 Lenox av
Ludden, James M., 233 W. 120th st
Ludlow, Ogden Curtis, 234 W. 135th st
Lumbard, Joseph Edward, Graham Court, Seventh av. and 116th st
Lusk, Thurston G., 121 E. 40th st
Lustgarten, Sigmund, 656 Madison av
Lyman, James W., 424 W. 34th st
Lynch, John B., 148 W. 22d st
Lynch, P. J., 216 E. 13th st
Lynde, George S., 326 W. 45th st
Lyon, Jonathan F., 231 E. 116th st
Lyons, D. Stanley, 223 E. 30th st
McAuliffe, Denis A., 310 E. 57th st
McAuliffe, George B., 57 E. 65th st
McBarron, John D., 872 West End av
McBurney, Charles, 28 W. 37th st
McCabe, Thomas, 335 2d av
McCallum, Samuel, 73 W. 127th st
McCastline, Robert, 108 E. 81st st
McClellan, E. S., Saranac Lake
McCosh, Andrew J., 16 E. 54th st
McCourt, Peter J., 233 W. 23d st
McCracken, William, 305 E. 18th st
McCreery, Forbes R., 123 E. 40th st
McCroskery, John, 371 Manhattan av
McCullagh, Samuel, 68 W. 38th st
McDonald, Dennis J., 137 E. 43d st
McFarland, Warren C., 360 W. 51st st
McGinnis, E. L. H., 202 W. 86th st
McGowan, J. P., 20 E. 29th st
McGrath, John J., 841 Lexington av
McInerny, J. H., 136 Manhattan av
McKay, William, 311 W. 23d st
McKenley, William H., 245 W. 30th st
McKernon, James F., 62 W. 52d st
McKim, W. Duncan, at present in Europe
McLaury, Frank Harold, 244 W. 42d st
McLean, Malcolm, 29 E. 126th st
McMahon, J. B., 325 Lexington av
McMannis, William T., 320 W. 45th st
McMaster, N. G., 63 E. 56th st
McMurdy, William S., 253 W. 70th st
McMurtry, Charles Wood, 812 Fifth av
McNulty, John J., 364 W. 35th st
McNutt, Julia G., 265 Lexington av
McNutt, Sarah J., 265 Lexington av
McSweeny, Daniel E., 129 E. 29th st

McWilliams, Clarence A., 50 E. 53d st
McWilliams, William Henry, 16 W. 96th st
Maass, Fritz, 691 Lexington av
Mabbott, J. Milton, 19 Fifth av
Macadam, Henry Graham, 325 W. 55th st
Macauley, Henri de Bournonville, 24 Oliver st
MacClymont, DeWitt Clinton, Bell. Hosp.
Macdonald, A. E., Med. Supt. Manhattan State Hosp., East, Ward's Island
Macdonald, Belle J., 346 W. 30th st
Macdonald, Carlos F., 85 Madison av
Macgregor, Robert Stuart, 338 W. 46th st
Machale, Ferdinand S., 317 E. 87th st
Macintyre, Burnett C., 146 W. 12th st
MacKee, George Miller, 119 W. 21st st
Mackie, D. H., 351 W. 56th st
Mac Nider, James Wright, 64 W. 56th st
Macphatter, Neil, 12 W. 40th st
Mac Phee, John Joseph, 367 W. 123d st
MacPherson, Duncan, 317 W. 119th st
Macy, Wm. Austin, Med. Supt. Willard State Hosp., Willard, N. Y.
Maddox, Albert Sidney, Madison av and 56th st "The Essex."
Magie, David, 7 W. 43d st
Magness, Frank Hosmer, Manhattan State Hosp., Central Islip, L. I.
Maher, J. J. E., 221 W. 23d st
Maier, Otto, 316 E. 18th st
Maisch, Charles O., 125 E. 24th st
Malcolm, Percy Edwin Dunlop, 118 Madison av
Mallett, E. Pierre, "The Dorilton," Broadway and 71st st
Mallett, George H., 144 W. 71st st
Mandlebaum, F. S., 1300 Madison av
Manges, Morris, 941 Madison av
Manley, Thomas H., 115 W. 49th st
Mannheimer, George, 1001 Madison av
Manson, Charles Albert, 2 W. 94th st
Markiewicz, Marcus, 1539 Madison av
Markoe, Francis H., 15 E. 49th st
Marple, Wilbur B., 35 W. 53d st
Marshall, David Trumbull, 33 W. 12th st
Martin, J., 437 W. 47th st
Martin, T. Dwight, 528 E. 161st st
Martus, Siegfried, 32 W. 114th st
Marvin, David M., 12 W. 130th st
Marx, S., 947 Madison av
Mason, E. Gaillard, 213 W. 79th st
Mason, William B., 62 W. 52d st
Mathews, Frank S., 62 W. 50th st
Matthews, George H., 1992 Lexington av
Mattoli, Agostino, 300 E. 107th st
Mauch, Joseph B., 238 W. 38th st
Maurer, George E., 158 E. 61st st
May, Calvin S., 57th st and Seventh av
May, Charles H., 698 Madison av
Mayer, Abraham, 40 E. 60th st
Mayer, Albert D., 241 E. 86th st
Mayer, Emil, 25 E. 77th st
Maynard, A. W., 308 W. 45th st
Maynard, W. H., 207 W. 55th st
Meacham, Leslie J., 69 W. 48th st
Meara, Frank Sherman, 400 W. End av
Medina, Jose L., 18 W. 32d st
Meding, Charles B., 157 W. 123d st
Meehan, James T., 234 E. 74th st
Meierhof, E. L., 1140 Madison av
Meirowitz, Philip, 202 W. 131st st
Meltzer, Samuel J., 166 W. 126th st
Meltzer, Victor, 177 W. 126th
Mendelson, Walter, 159 W. 74th st
Mennen, John H., 4 E. 8th st
Merchant, Francis D., Batopilas, Mexico
Merrill, Estelle C., 438 W. 116th st
Mesereau, William J., 152 E. 34th st
Messemer, Edward J., 144 Second av
Meyer, Alfred, 801 Madison av
Meyer, Alfred Edward, 102 W. 93d st
Meyer, Leo B., 60 W. 129th st
Meyer, Willy, 700 Madison av
Meyers, Geo., 47 E. 61st st
Mial, Leonidas L., 139 W. 12th st
Michaelis, Alfred, 173 E. 95th st
Michaelis, Ludwig M., 1090 Lexington av
Middleton, John Izard, 60 W. 51st st
Milbank, Robert, 154 W. 48th st
Mildenberg, Victor, 1469 Lexington av
Milleg, Augustus, 147 E. 47th st
Miller, Elmer A., 1187 Madison av
Miller, Edward, 231 E. 60th st
Miller, Frank E., 22 W. 31st st
Miller, George N., 811 Madison av
Miller, J. Preston, 500 W. 149th st
Mills, Jackson M., 695 Madison av., near 62d st
Millspaugh, Louis C., 117 W. 81st st
Minden, Simeon B., 230 E. 69th st
Minor, John C., 65 W. 73d st
Minor, S. Carrington, 850 E. 165th st
Mislig, Michael, 330 E. 72d st

Mitchell-Kydd, Mary, cor. Sip and Tonnele avs., J. C. Hts.
Mittelstaedt, Charles B., 86 University pl
Mittendorf, William F., 140 Madison av.
Mittendorf, William Keith, 140 Madison av
Moak, Charles H., 201 W. 121st st
Moeller, Henry, 341 W. 57th st
Molner, Nathan, 464 Grand st
Monell, S. H., 47 W. 27th st
Mooney, Charles James, 44 E. 63d st
Mooney, Henry W., 26 W. 121st st
Moore, James Basil, 142 E. 40th st
Moore, John Frederick, 156 W. 94th st
Moore, William, 320 Broadway
Moore, William Oliver, 27 E. 30th st
Moorhead, John J., 101 Manhattan av
Moorhead, Joseph, 310 Second av
Moran, James, 152 W. 57th st
Morje, Benjamin, 116 E. 73d st
Morrill, J. Lee, 67 E. 79th st
Morris, George F., 161 W. 87th st
Morris, Lewis, 219 W. 22d st
Morris, Louis, 159 E. 56th st
Morris, Robert S., 152 E. 71st st
Morris, Robert T., 58 W. 56th st
Morrissey, John J., 50 E. 126th st
Morrow, Prince A., 66 W. 40th st
Mortimer, William Golden, 107 W. 72d st
Morton, Bowditch, University Club
Morton, William James, 19 E. 28th st
Moschcowitz, Alexis V., 925 Madison av
Moseley, Henry Perkins, 939 Madison av
Moseley, William B., Manhattan State Hosp., West, Ward's Island
Moser, George J., 283 E. 10th st
Moses, Mortimer A., 73 Worster st
Mowry, Eugene C., 355 W. 42d st
Muellenbach, Robert P., 232 E. 10th st
Muir, Amasa P., 204 W. 133d st
Muller, Alfons, 334 E. 84th st
Muller, Emil Alfred, 345 Pleasant av
Muller, Henry W., 427 E. 85th st
Muller, Richard W., 147 E. 18th st
Mund, Adolph, 308 E. 62d st
Mund, Carl, 962 Lexington av
Mundorff, George Theodore, 304 Second av
Munn, John P., 18 W. 58th st
Munroe, George E., 43 E. 33d st
Murphy, Deas, 444 Park av
Murray, Charles Basil, 307 E. 79th st
Murray, Francis J., 28 W. 61st st
Murray, Grace Peckham, "The Langham," cor. Fifth av and 52d st
Murray, Robert A., 112 W. 80th st
Murtland, Samuel, 14 W. 47th st
Musgrave, Christopher J., 65 Monroe st
Myers, Howard G., 175 W. 73d st
Myers, Lotta Wright, 180 W. 74th st
Myers, Thad. Halsted, 54 W. 50th st
Myles, Robert C., 46 W. 38th st
Nagle, J. Darwin, 61 W. 35th st
Nagle, John T., 163 W. 126th st
Nammack, Charles E., 42 E. 29th st
Nash, George W., Kingsbridge
Nash, Miles Henry, 217 W. 43d st
Needham, George Gordon, 218 E. 19th st
Neff, Lewis Knode, 1213 Park av
Neftel, William B., 16 E. 48th st
Neil, James, 74 W. 131st st
Nelson, Wolfred, Astor House
Nesbitt, John H., 212 W. 45th st
Neufield, Albert W., 113 E. 116th st
Neuhaus, George Emile, 162 W. 93d st
Neuhof, Selian, 235 E. 86th st
Neumann, Louis, 459 W. 24th st
Newcomb, Albert S., 206 W. 71st st
Newcomb, James E., 118 W. 69th st
Nichols, James Allen, 55 W. 75th st
Nicolai, Curt. E. H., 81 W. 119th st
Nicoll, Henry D., 51 E. 57th st
Nicoll, Matthias, Jr., 124 E. 60th st
Nilsen, J. R., 69 W. 50th st
Nisbet, J. Douglas, 10 E. 43d st
Noble, John G., 304 W. 77th st
Nordemann, Felix, 210 E. 60th st
Norrie, V. H., 21 W. 37th st
Norris, Henry S., 10 W. 49th st
North, James H., Jr., 346 Broadway
Northrup, Wm. P., 57 E. 79th st
Norton, Nathaniel Read, 107 E. 70th st
Noyes, William Bradbury, 28 W. 61st st
Nye, Frances Asenath, 1938 Lexington av
Oastler, Frank R., 126 W. 59th st
Oberle, John H., 1758 Bathgate av
O'Brien, John M., 308 W. 34th st
O'Brien, M. C., 161 W. 122d st
O'Connor, John H., 47 W. 16th st
Ogilvy, Charles, 48 E. 31st st
O'Neill, C. H. F., 161 W. 97th st
Opdyke, Ralph, 137 W. 69th st
Oppenheimer, Seymour, 45 E. 60th st
Osborne, Lawrence J., 246 W. 43d st
Osgood, Alfred Townsend, 77 W. 50th st
Otis, William K., 5 W. 50th st
Outerbridge, Paul E., 27 W. 74th st

Owen, Henry E., 40 W. 56th st
Owsley, Henry F., 259 Amity, Flushing, L. I.
Oyler, W. H., 216 W. 124th st
Packard, Charles W., 447 Park av
Packer, Flavius, Bellevue Hosp.
Page, Kingman B., 24 W. 120th st
Painter, Henry McM., 62 W. 55th st
Palmer, Edmund Janes, 103 W. 86th st
Park, Wm. Hallock, 315 W. 76th st
Parker, Frank J., 49 W. 50th st
Parker, George M., 515 Madison av
Parodi, Teofilo, 127 E. 60th st
Parry, Angenette, 249 E. 32d st
Parsons, John, Kingsbridge
Parsons, Ralph L., 21 E. 44th st
Parsons, Ralph W., 21 E. 44th st
Partridge, Edward L., 19 Fifth av
Pascal, Henry S., 318 W. 52d st
Patterson, F. N., 21 E. 44th st
Patterson, Henry Stewart, 51 W. 58th st
Payn, Abbott Smith, 545 W. 148th st
Payne, Sanders McAllister, 542 Fifth av
Peck, Charles Howard, 37 W. 48th st
Peck, Edward Sprague, 53 W. 50th st
Pedersen, James, 20 E. 46th st
Peet, Edward W., 348 Central Park, West
Peirce, Henry T., 272 Lenox av
Perkins, Edward W., 224 W. 131st st
Perry, Ellis Banning, 102 W. 48th st
Perry, John Gardner, 48 E. 34th st
Peterson, Edward Wadsworth, 12 W. 40th st
Peterson, Frederick, 4 W. 50th st
Peterson, J. Smith, 112 E. 85th st
Pettit, Louis Clark, Ward's Island
Pfeiffer, Alfred G., 230 E. 53d st
Pfeiffer, Joseph, 130 E. 62d st
Phelps, Frank V., 231 Lexington av
Phelps, Oscar S., Battle Creek, Mich.
Phillips, Charles Ernestus, 45 W. 119th st
Phillips, Wendell C., 40 W. 47th st
Piffard, Henry G., 256 W. 57th st
Pinckney, S. G. Courtney, Atlanta, Ga.
Pisani, Antonio, 38 New Bowen st
Pisek, Godfrey Roger, 250 E. 72d st
Pisko, Edward, 151 E. 78th st
Plimpton, W. O., 12 E. 28th st
Polk, William M., 7 E. 36th st
Pollitzer, Sigmund, 64 E. 58th st
Ponce de Leon, N. J., 146 W. 65th st
Pool, Eugene H, 57 W. 45th st
Pooley, Thomas R., 107 Madison av
Poore, Charles T., 43 W. 53d st
Porter, W. Evelyn, 50 W. 33d st
Porter, William H., "The Strathmore," Broadway, cor. 52d st
Potter, E. Styles, 214 W. 92d st
Potter, La Forest, 1048 Fifth av
Potter, Nathaniel Bowditch, 9 W. 35th st
Pound, W. H., West Farms road, Westchester, N. Y.
Powell, Seneca D., 12 W. 40th st
Power, James Francis, 230 W. 34th st
Prellwitz, Otto Emil, 304 E. 23d st
Pretlow, Robert Henley, 317 W. 18th st
Prevost, Charles A. Wilson, 111 Madison av
Price, John Adolph, 117 Second av
Pritchard, Wm. Broaddus, 105 W. 73d st
Pryor, William Rice, 6 W. 84th st
Pulley, W. J., 945 Madison av
Purdy, Alfred E. M., 304 Madison av
Purdy, Harry Lavinton, 116 E. 74th st
Purdy, Harry R., 149 Lexington av
Putnam, Charles R. L., 121 E. 38th st
Putzel, Leopold, 13 E. 57th st
Quackenbos, Henry F., 159 W. 88th st
Quimby, Charles E., 44 W. 36th st
Quinlan, Francis J., 33 W. 38th st
Quintard, Edward, 145 W. 58th st
Rabenstein, Hans, 317 E. 6th st
Rabinowitsch, Hirsch, 168 Madison st
Rae, John Broadfoot, 668 E. 157th st
Rafel, Henry, 106 W. 87th st
Ramsdell, E. Benjamin, 581 Lexington av
Ramsay, George D., 30 Kay st., Newport, R. I.
Ranney, Ambrose L., 345 Madison av
Ransom, Charles C., 66 W. 49th st
Rapp, Samuel, 134 E. 79th st
Rau, Leonard S., 161 W. 74th st
Rautenberg, Godfrey W., 335 E. 87th st
Reese, Robert Grigg, 58 E. 25th st
Reid, Adrian Y., 104 Lexington av
Reid, John J., 853 Lexington av
Reiley, I. H., 459 W. 22d st
Reinthaler, Jonas E., 72 E. 81st st
Reis, Herman Louis, 215 E. 11th st
Renn, Pius, 176 E. 95th st
Requa, Mary Augusta, 55 W. 52d st
Reynolds, Walter Seymour, 54 W. 94th st
Rhein, Meyer L., 38 E. 61st st
Rice, Clarence C., 123 E. 19th st
Rice, Lucien G., Elk, New Mexico
Richard, Montrose R., 77 E. 116th st
Richardson, Alfonso A., 124 W. 111th st

Richardson, Charles H., 417 Lexington av
Richardson, Jennie M., Ilion, N. Y.
Richter, William, 235 E. 19th st
Ripperger, Albert A., 50 W. 130th st
Ritter, Joseph Cambridge, 162 Waverly pl
Rives, William Cabell, 1723 I st., Washington, D. C.
Robbins, Jane Elizabeth, 446 E. 72d st
Robertson, Joseph A., 58 W. 33d st
Robertson, Oswald Dundas Farquharson, 71 E. 82d st
Robeson, F. T., 325 Central Park, West
Robie, John W., 184 W. 82d st
Robinson, Alexander L., 59 W. 44th st
Robinson, A. R., 159 W. 49th st
Robinson, Beverley, 42 W. 37th st
Robinson, Fred C., 221 W. 11th st., near Greenwich av
Robinson, Wm. J., 119 E. 128th st
Rockwell, A. D., 25 E. 44th st
Rockwell, William H., Jr., 141 W. 76th st
Rodenstein, Louis A., 561 W. 152d st
Rodgers, William A., 816 Lexington av
Rodman, Harry, 158 E. 116th st
Roediger, Traugott, F. M., 312 W. 35th st
Rogers, Edward H., 518 W. 150th st
Rogers, Henry Alexander, 1443 Lexington av
Rogers, John, 102 E. 30th st
Rogers, Oscar H., 346 Broadway
Rogers, Willard H., 225 W. 22d st
Romaine, De Witt C., 473 Hudson st
Romm, Maxim D., 306 E. 15th st
Roos, Lester Laurens, 233 W. 113th st
Roosa, D. B. St. John, 20 E. 30th st
Rose, Achilles, 126 E. 29th st
Rosenbaum, Louis, 1458 Lexington av
Rosenberg, E., 138 W. 85th st
Rosenberg, Julius, 1003 Madison av
Rosenberg, Leopold, 112 W. 128th st
Rosenberg, Max, 128 E. 70th st
Rosenheim, Paul Joseph, 122 E. 55th st
Rosenthal, Max, 120 E. 80th st
Rosenwasser, Charles Adolph, 1242 Madison av
Ross, William, 354 W. 24th st
Roth, Henry, 663 E. 140th st
Rottenberg, Ignatz Morvay, 280 2d st
Rowe, John T. W., Manhattan State Hosp., Ward's Island
Rowland, Henry C., 120 E. 34th st
Rudisch, Julius, 39 E. 63d st
Rundquist, Emil Albin, 71 W. 55th st
Rupp, Adolph, 359 W. 34th st
Ruppe, Ernest F., 427 W. 47th st
Rushmore, Mary D., 102 E. 26th st
Russell, York, 317 W. 36th st
Ryder, George Hope, 515 Madison av
Ryniker, Henry J., 155 E. 48th st
Sabel, Stanley Owen, "Windsor Hotel," Orange, N. J.
Sachs, B., 21 E. 65th st
Salisbury, T. B., 250 W. 57th st
Sanders, Charles Walton, 53 E. 53d st
Sarlabous, Emile J., 127 W. 22d st
Satterlee, George Reese, 60 E. 78th st
Satterthwaite, Thomas E., 7 E. 80th st
Saunders, R. H., 131 E. 61st st
Savage, Henry Burnham, 308 W. 59th st
Savage, Watson Lewis, 308 W. 59th st
Savidge, Eugene Coleman, 66 W. 50th st
Savini, Carlo, 206 E. 17th st
Saxe, G. A. DeSantos, 75 W. 55th st
Saxl, Josef, 244 E. 72d st
Sayre, Reginald H., 9 E. 45th st
Schapringer, A., 926 Madison av
Scharlau, Barnim, abroad permanently
Scheider, Julius, 135 E. 74th st
Schelpert, John Wm., 10 W. 93d st
Schiff, Herman James, 117 E. 79th st
Schimpf, Henry William, 404 W. 34th st
Schlegel, Gustavus, 151 W. 91st st
Schlereth, George, New Rochelle
Schlereth, Mathilda M., New Rochelle
Schley, Fayette E., 473 West End av., cor. 83d st
Schley, Winfield Scott, 77 W. 50th st
Schnecker, William Augustus Muhlenberg, 596 Lexington av
Schoenberg, Marcus T., 101 E. 4th st
Schoenenberger, Frederick J., 344 W. 29th st
Schoeps, George, 206 W. 122d st
Schöney, Lazarus, 143 W. 142d st
Schoonover, Warren, 115 E. 59th st
Schoonover, Warren, Jr., 115 E. 59th st
Schott, Max, 162 E. 74th st
Schram, Charles, 52 E. 82d st
Schroeder, Henry H., 32 Nassau st
Schultz, Ernest C., 103 W. 77th st
Schultze, Otto H., 1109 Madison av
Schwartz, Martin, 129 E. 90th st
Schwedler, Edward F., 312 W. 34th st

Schwerd, Max Joseph, 13 W. 120th st
Schwyzer, Fritz, 54 E. 58th st
Scofield, Richard Jessup, 349 W. 14th st
Scott, Elmer A., 931 West End av
Scott, George, Germantown, Pa.
Scott, Henry, 216 W. 121st st
Scratchley, Francis Arthur, 110 E. 25th st
Seabrook, Harry Hartshorne, 118 E. 72d st
Seaman, Louis L., 18 W. 31st st
Seamans, William S., 120 Broadway
Seeligman, Gustav, 53 E. 72d st
Seibert, August, 114 E. 57th st
Sellew, Frederick S., 61 E. 79th st
Seltzer, Isaac M., 104 W. 94th st
Senftenberg, Benj. F., 101 E. 95th st
Senftner, Herman Francis, 148 W. 122d st
Seward, W. M., 55 E. 86th st
Sewell, Walter D., Murray Hill Hotel
Shaffer, Newton M., 28 E. 38th st
Shannon, John Rowlands, 20 W. 35th st
Shannon, Wm., 130 W. 81st st
Sharp, John Clarence, 62 W. 46th st
Shaw, Frank, E. E., 411 W. 145th st
Sheedy, Bryan De Forest, 10 W. 46th st
Shelby, Edmund P., 116 W. 74th st
Sherman, A. Josephine, 58 E. 75th st., Sanatorium
Sherwood, Milton See, 871 St. Nicholas av., bet. 153d and 154th sts
Shine, Francis Eppes, 126 W. 48th st
Shively, Henry Laurence, 303 Amsterdam av
Shotwell, John Byron, 328 W. 46th st
Shrady, George F., 8 E. 66th st
Shrier, Albert Franklin, 110 E. 84th st
Shultz, P. David, 223 W. 48th st
Siegelstein, Pierre A., 85 Rivington st
Silver, H. M., 5 E. 43d st
Silver, Lewis M., 103 W. 72d st
Simmons, Charles E., 762 Madison av
Simmons, John Gorse, Westchester av., bet. Aves. C and D
Simpson, William Kelly, 952 Lexington av
Simpson, William Payne, 908 St. Nicholas av., cor. 155th st
Smiley, Alton L., Manhattan State Hospital, East, Ward's Island
Smith, A. Alexander, 18 W. 51st st
Smith, Alfred K., 51 Manhattan av
Smith, Andrew Heermance, 18 E. 46th st
Smith, Charles, 71 E. Houston st
Smith, Edward Franklin, 324 W. 46th st
Smith, Ernest Ellsworth, 26 E. 29th st
Smith, Francis Eugene, 306 W. 55th st
Smith, Harmon, 122 E. 34th st
Smith, J. Gardner, 21 W. 122d st
Smith, Samuel Wesley, 66 Madison av
Sobel, Jacob, 1828 Madison av
Solley, Fred Palmer, 33 W. 53d st
Solomonides, John Joseph, 40 E. 10th st
Sondern, Frederic E., 200 W. 56th st
Sondern, Paul Frederick, 19 E. 38th st
Sour, Bernard, 68 W. 97th st
Southworth, Thomas S., 807 Mad. av
Spalding, Alfred M., 419 W. 145th st
Spalding, George A., 248 Lenox av
Spalding, Warren C., "Hotel Winthrop," 7th av and 125th st
Spann, J. H., 102 W. 74th st
Spellman, Dwight Seymour, Ass't. Phys. Manhattan State Hospital, Ward's Island
Spence, Arnot, 70 W. 71st st
Spence, D. Benham, 615 W. 181st st
Spier, Oscar A., 179 E. 108th st
Spiller, William H., 307 2d av
Spitzer, Henry, 78 E. 92d st
Spitzka, Edward C., 66 E. 73d st
Spooner, Henry Garrettson, 25 W. 44th st
Sprague, Homer B., 1383 Lexington av
Squier, J Bentley, 20 E. 46th st
Stark, Henry S., 156 E. 72d st
Starr, Moses Allen, 5 W. 54th st
Stearns, Henry S., 45 W. 58th st
Stedman, Thomas L., 1425 Broadway
Steel, George E., 124 W. 119th st
Steese, Edwin Sturtevant, 46 E. 57th st
Steffens, William, 312 E. 120th st
Stein, Richard, 79 E. 79th st
Steinach, William, 134 E. 18th st
Steinberger, Victor, 350 E. 84th st
Stella, Antonio, 365 Broome st
Stephens, Franklin Moore, 71 W. 49th st
Sterling, Kate L. S., "The Lowell," 120th st and Amsterdam av
Stern, Henrich, 56 E. 76th st
Stern, Maxmilian, 141 W. 43d st
Sternberg, Frederick Alexander, 414 E. 50th st
Sternberger, Edwin, 43 E. 60th st
Steurer, John A., 78 W. 47th st
Stevens, George T., 22 E. 46th st

Stevens, William, 70 W. 52d st
Stevenson, Clinton, 1227 Lexington av
Stevenson, Thomas, 220 W. 105th st
Stewart, Douglas Hunt, 111 W. 64th st
Stiefel, Benjamin Wolf, 106 E. 81st st
Stieglitz, Leopold, 60 E. 65th st
Stillwell, John E., 9 W. 49th st
Stimson, Charles W., 68 W. 40th st
Stimson, Daniel M., 11 W. 17th st
Stimson, L. A., 34 E. 33d st
Stone, J. B., 70 W. 52d st
Stone, William F., 340 W. 57th st
Stone, Wm. Metcalfe, 63 Jamaica av., Flushing
Stone, William S., 1730 Broadway, cor. 55th st
Stowell, William L., 160 W. 73d st
Straus, Ludwig, Hotel New Amsterdam
Straus, Rosa Welt, 38 W. 84th st
Strong, Cyrus John, 60 W. 75th st
Stubbert, J. Edward, 25 E. 45th st
Stubenbord, William, 219 W. 38th st
Sturgis, Frederic Russell, 16 W. 32d st
Sturmdorf, Arnold, 51 W. 74th st
Swan, Albert T., 125 E. 55th st
Swasey, John H., 34 E. 28th st
Sweeney, Thompson Tyler, 158 E. 61st st
Swift, Edwin E., 112 W. 81st st
Swift, George Montague, 20 W. 55th st
Swift, William J., 40 E. 30th st
Swinburne, George Knowles, 64 E. 56th st
Symonds, Brandreth, 34 Nassau st
Taddiken, Paul Gerald, L. I. State Hosp., Kings Park, L. I.
Taft, Robert Mac Lean, 223 W. 57th st
Talbot, Robert Bancker, 37 W. 68th st
Talmey, Bernhard S., 6 W. 123d st
Talmey, Max, 150 E. 81st st
Talson, George W., 129 W. 95th st
Taltavall, William Allan, 2137 Seventh av
Tandlich, Samuel, 328 E. 52d st
Tannenbaum, Julius, 56 W. 112th st
Tannenbaum, Simon, 349 E. 83d st
Tansley, J. Oscroft, 28 W. 43d st
Taylor, Fielding Lewis, 173 W. 73d st
Taylor, George Ash, 46 W. 36th st
Taylor, Harry Neafie, 35 E. 27th st
Taylor, Henry Ling, 125 W. 58th st
Taylor, Howard Canning, 24 W. 50th st
Taylor, Robert W., 40 W. 21st st
Taylor, William Wallace, 75 E. 116th st
Terrell, Allen Greenleaf, 406 W. 57th st
Teschner, Jacob, 134 E. 61st st
Thacher, John Seymour, 839 Madison av
Thelberg, John, 45 W. 43d st
Thelberg, Martin A. H., 205 W. 105th st
Theobald, Carl, 230 E. 12th st
Theyson, Robert H., 104 E. 10th st
Thomas, Allen M., 45 W. 54th st
Thomas, William S., 68 W. 52d st
Thompson, Edmund Burke, 316 W. 105th st
Thompson, Edward Middleton, 221 W. 57th st
Thompson, George S., 405 E. 17th st., Brooklyn
Thompson, Von Beverhout, 107 E. 60th st
Thompson, W. Gilman, 34 E. 31st st
Thomson, C. H., 202 N. 14th av., Seattle, Wash.
Thomson, Edgar Steiner, 19 E. 44th st
Thomson, William H., 23 E. 47th st
Thomson, W. Ross, 10 W. 128th st
Thornley, Josiah Payne, "Hotel Ansonia," B'way and 74th st
Tillinghast, Joseph Reynolds, Jr., Colorado Spg's., Col.
Tilton, Benjamin T., 49 E. 53d st
Titus, Edward C., 117 W. 11th st
Tobias, Leo, 326 E. 52d st
Tompkins, Byron V., 206 W. 86th st
Tompkins, Walsten M., 49 W. 38th st
Torek, Franz J. A., 59 E. 60th st
Tosti, Gustavo, 692 Madison av
Tousey, Ralph, 259 W. 139th st
Tousey, Sinclair, 103 W. 76th st
Tovey, David William, 2400 7th av
Townsend, Frederick M., 45 W. 32d st
Townsend, Theodore Irving, Utica State Hosp., Utica, N. Y.
Townsend, Wisner R., 125 W. 58th st
Tracy, E. Clark, 27 E. 126th st
Tranchida, Corrado, 106 E. 18th st
Traub, John Emil, 228 W. 75th st
Travell, John Willard, 27 E. 11th st
Travis, Alexander H., 130 W. 82d st
Trenwith, Walter Day, 140 W. 94th st
Trimble, William B., 56 E. 25th st
Tuck, Henry, 346 Broadway
Tucker, C. P., 43 W. 26th st

Tuffs, Edward G., 30 Oliver st
Tull, Edward Emory, 119 W. 80th st
Tully, Marcus E., 137 W. 76th st
Tupper, Benjamin Randall, 246 W. 51st st
Turrell, Guy Hanford, 1787 Washington av
Tuthill, Theodore K., 319 W. 18th st
Tuttle, George Montgomery, 38 W. 52d st
Tuttle, James P., 42 W. 50th st
Tynberg, Sigmund, 1329 Madison av
Tyson, Henry H., 47 W. 51st st
Unger, James Samuel, 320 E. 120th st
Valadier, Charles A., 297 Central Park, West
Valentine, Ferd. C., 31 W. 61st st
Valentine, William A., 34 W. 38th st
Valk, Francis, 164 E. 61st st
Vandegrift, George M., 515 Grand st
Van Derlyn, J. Dubois, 174 E. 71st st
Van Der Poel, John, 36 W. 39th st
Van Der Poel, S. O., 346 Broadway
Vanderpoel, Waldron B., 106 E. 24th st
Van Dyck, Clinton DeWitt, 47 W. 93d st
Van Fleet, Frank, 60 E. 77th st
Van Fleet, Milton E., 2d, Williamsbridge
Van Loan, James Casper Plimpton, 346 W. 33d st
Van Santvoord, Richard, 10 W. 122d st
Vantine, Jefferson Brockner, 127 W. 11th st
Van Valzah, W. W., 10 E. 43d st
Van Vredenbergh, William T., New Brighton, S. I.
Van Wert, Charles, 115 W. 97th st
Vedder, Maus R., 35 E. 62d st
Vedin, Augusta, 224 E. 15th st
Vincent, Ludger C., 103 W. 77th st
Vincent, Wesley Groove, 74 W. 82d st
Vineberg, Hiram N., 751 Madison av
Virden, J. E., Ferris pl., Westchester
Voislawsky, Antoine P., 128 W. 59th st
Volkenberg, Albert, 315 E. 17th st
Von Grimm, Andrew, 244 E. 13th st., bet. 2d and 3d avs
Von Raitz, F., 1 W. 102d st
Von Ramdohr, C. A., 45 Irving pl
Voorhees, James Ditmars, 150 W. 59th st
Vosburgh, Arthur S., 40 W. 88th st
Wachenheim, Frederick L., 105 E. 80th st
Wainright, Charles F., 47 E. 58th st
Wakefield, Homer, 151 W. 76th st
Waldo, Ralph, 68 W. 50th st
Walker, Charles H., 105 W. 77th st
Walker, D. Ernest, 254 W. 45th st
Walker, Emma Elizabeth, "Martha Washington," 29 E. 29th st
Walker, Henry Freeman, 18 W. 55th st
Walker, John B., 33 E. 33d st
Wallace, Charlton, 391 West End av
Wallain, Samuel Spencer, 503 Fifth av
Walsh, Simon J., 25 E. 128th st
Walter, Josephine, 61 W. 74th st
Walton, Luis P., 73 W. 50th st
Ward, Edwin F., 15 W. 96th st
Ward, Freeman Ford, 6 E. 58th st
Ward, George Gray, Jr., 230 W. 59th st
Ward, Thomas Logan, 278 W. 127th st
Ware, Edward J., 121 W. 93d st
Ware, Martin W., 1198 Lexington av
Warford, Geo. T., 132 W. 82d st
Wark, David, 315 W. 48th st
Warner, Everett S., 56 W 56th st
Warren, John, 106 E. 29th st
Warren, John S., 150 W. 48th st
Warsaw, M. Claudius, 242 W. 45th st
Warwick, Hill Sloane, 254 W. 121st st
Waterman, Jerome Hilton, 50 W. 51st st
Waters, Bertram Howard, 22 E. 46th st
Waters, John Sherreaf, 48 W. 83d st
Watkins, Robert L., Astor Court, 20 W. 34th st
Watts, Robert, 45 W. 36th st
Watts, Robert, Jr., 111 E. 35th st
Webb, Walter D., Ass't. Surg. U. S. Army, War Dept., Washington, D. C.
Weber, Leonard, 25 W. 46th st
Weber, Leonard G., 624 Lexington av
Webster, David, 327 Madison av
Weed, Edgar T., 117 W. 121st st
Weeks, John E., 46 E. 57th st
Weinstein, Harris, 254 E. B'way
Weiss, Ludwig, 77 E. 91st st
Weisse, Faneuil D., 46 W. 20th st
Welker, Franklin, 344 W. 145th st
Welles, Charles Stuart, 9 Roland Gardens, London, S. W.
Wells, Brooks H., 34 W. 45th st
Wells, Edward H., 141 E. 89th st
Welt-Kakels, Sara, 71 E. 66th st
Welzmiller, John, 42 E. 28th st
Welzmiller, Louis Reed, 264 W. 57th st
Wendt, Edmund Charles, 118 W. 79th st

Wenner, George, 352 W. 40th st
West, James Nephew, 71 W. 49th st
West, William Edward, 330 W. 85th st
Westcott, N. S., 159 W. 12th st
Westerfield, William, 109 W. 123d st
Westermann, Julius T., 104 Lexington av
Weston, Albert T., 226 Central Park, West
Weston, William H., 400 W. 22d st
Wetmore, Geo. T., 36 W. 35th st
Wharton, John Cambridge, 102 W. 89th st
Wheeler, Lyman Hall, 31 W. 84th st
Wheelock, George G., 75 Park av
Wheelock, William E., Morristown, N. J.
White, Charles Bell, 405 W. End av
White, Granville Moss, 272 W. 77th st
White, John Blake, 1013 Madison av
White, Octavius A., 41 E. 78th st
Whitehouse, Henry H., 24 W. 36th st
Whiting, Charles Allen, 70 W. 47th st
Whiting, Fred., 47 W. 50th st
Whiting, James Raynor, Jr., 120 W. 55th st
Whitman, Royal, 283 Lexington av., near 36th st
Whitmyer, John Franklin, 130 W. 70th st
Whitney, Charles Alvano, 120 E. 34th st
Wickliffe, John Wakefield, Manhattan State Hosp., Ward's Island
Wiener, Alfred, 113 W. 77th st
Wiener, Joseph, 1046 Fifth av
Wiener, Joseph, Jr., 1001 Madison av., near 77th st
Wiener, Richard G., 48 E. 65th st
Wiesner, Daniel H., 162 E. 46th st
Wilcox, Reynold Webb, 679 Madison av
Wilkens, Ernst A. W., 284 Alexander av
Wilkin, William Porter, 60 W. 51st st
Willard, Thomas H, 1 Madison av
Williams, Charles Mallory, 531 Madison av
Williams, E. Ellsworth, 156 Madison av
Williams, Linsly R, 839 Madison av
Williams, Mark H., 230 W. 135th st
Williams, William Robert, "San Remo," 74th st and Central Park, West
Williamson, Edward Lincoln, 163 W. 74th st
Wilsey, Orville Jay, 143 E. 37th st
Wilson, Frederic Newhall, 106 W. 47th st
Wilson, Robert J., 342 W. 71st st
Winters, Joseph E., 25 W. 37th st
Wischnewetzky, Lazare, 334 Fifth av
Wise, Peter M., 1135 Broadway
Witson, Albert J., 318 W. 126th st
Wohlfarst, Justin, 119 W. 129th st
Wolbarth, Abraham Leo, 187 Henry st
Wolf, Henry J., 131 W. 126th st
Wolfe, Augustin A., 603 Lexington av
Wolff, Julius, 52 E. 64th st
Wolper, Max, 217 E. Broadway
Wood, Francis Carter, "The Osborne," 205 W. 57th st
Wood, Halsey Lathrop, 86 Madison av
Wood, Thomas D., 417 W. 118th st
Wood, William Benjamin, 33 W. 47th st
Woodend, William E., 58 W. 71st st
Woodman, John, 56 W. 56th st
Woodward, Julius Hayden, 58 W. 40th st
Woolsey, George, 117 E. 36th st
Worster, Willard Parker, 203 W. 81st st
Wright, Arthur Brownell, Manhattan State Hosp., Ward's Island
Wright, John P., 221 E. 82d st
Wyckoff, Peter B., 23 W. 57th st
Wylie, Robert Hawthorne, 36 W. 35th st
Wylie, W. Gill, 28 W. 40th st
Wynkoop, D. Woodbury, 128 Madison av
Wynkoop, Gerardus H., 128 Madison av
Yale, Leroy Milton, 432 Madison av
Yard, Andrew Benedict, 28 W. 105th st
Young, Clement C., 2043 Seventh av
Young, J. V. D., 60 W. 76th st
Youngling, George S., 453 W. 34th st
Zabriskie, Fred Templeton, 58 W. 47th st
Zeh, Adolph, 243 W. 52d st
Zemansky, A. P., 149 Broadway
Zweighaft, Bernard, 54 W. 71st st
Zwisohn, Lazarus W., 1085 Lexington av

Number of Members, 1728.

NEW YORK ACADEMY OF MEDICINE.

(Instituted January 6, 1847. Incorporated June 23, 1851.)

MEETINGS.—Stated, first and third Thursdays in every month, from the first Thursday in October to the first Thursday in June, inclusive, at 8 P. M., at 17 W. Forty-third street, New York. Library open free to the public from 10 A. M. to 6 P. M., to members from 10 A. M. to 10 P. M.; in summer, 10 A. M. to 6 P. M. Physicians in active practice for the three years preceding application, and residents of the State of New York, are eligible for Resident Fellowship. Surgeons in the U. S. Army, Navy, or Marine Hospital Service, and physicians residing in other states, are eligible for Non-resident Fellowship.

Officers. (1903.)

Andrew N. Smith, *President.*

Charles L. Dana, Virgil P. Gibney, M. Allen Starr, *Vice-Presidents.*

John H. Huddleston, *Recording Secretary.*
126 W. 85th street, New York City.

Louis Faugères Bishop, *Corresponding Secretary.*

Herman L. Collyer, *Treasurer.*

Trustees: Abraham Jacobi, *Chairman,* Joseph D. Bryant, Arthur M. Jacobus, Edward G. Janeway, A. Alexander Smith.

Reginald H. Sayre, *Treasurer for the Trustees.*

Committee on Admissions: J. Blake White, *Chairman.*

Committee on Library: Augustus Caillé, *Chairman.*

Mr. John S. Browne, *Resident Librarian.*

Executive Committee: Abraham Jacobi, *Chairman.*

Howard Van Sinderen, *Counsel to the Academy.*

MEDICAL SOCIETY OF THE COUNTY OF NIAGARA.

MEETINGS.—Annual, first Tuesday in June; semi-annual, first Tuesday in January; quarterly, first Tuesday in April and September.

Officers. (June, 1902.)

George H. Minard, *President.* J. H. Miller, *Vice-President.*
F. A. Crosby, *Secretary.* W. A. Scott, *Treasurer.*

Censors: M. S. Kittinger, J. H. Miller, F. J. Baker, H. A. Wilmot.
Committee on Hygiene: A. N. Moore, *Chairman,* F. J. Baker, M. S. Kittinger, Walter McChesney, M. H. Cole, T. B. Cosford.

Members.

F. J. Baker, Lockport
J. W. Bickford, Lockport
T. P. C. Brainard, North Tonawanda
C. E. Campbell, La Salle
H. S. Campbell, Niagara Falls
Wm. R. Campbell, Niagara Falls
Frederick T. Carmer, Rapids
A. L. Chapin, Niagara Falls
Clayton R. Clarke, Ransomville
M. H. Cole, Newfane
J. W. Corman, North Tonawanda
T. B. Cosford, Lockport
F. A. Crosby, Lockport
B. F. Dennis, Niagara Falls
W. L. Draper, Wilson
J. Glen Ernest, Gasport
W. J. Falkner, Youngstown
Frank Guillemont, Niagara Falls
J. B. Hartwell, Lockport
J. H. Helmer, Lockport
Thomas M. Heard, Jr., Ransomville
W. H. Hodson, Lockport
W. Q. Huggins, Sanborn
A. W. Jackson, Gasport
F. A. Kittinger, Lockport
M. S. Kittinger, Lockport
William Lane, Lockport
Allen T. Leonard, Tonawanda
W. H. Loomis, Lockport
Thomas J. McBlain, Niagara Falls
F. R. McBrien, Niagara Falls
Walter McChesney, Niagara Falls

A. A. McNamara, Lockport
H. H. Mayne, Lockport
J. H. Miller, Niagara Falls
George H. Minard, Lockport
A. N. Moore, Lockport
M. F. Mudge, Johnston Creek
C. N. Palmer, Lockport
Wm. H. Potter, Niagara Falls
W. B. Rice, Lockport
E. N. S. Ringueberg, Lockport
W. A. Scott, Niagara Falls
Edwin Shoemaker, Charlotteville
W. S. Town, North Ridge
H. A. Wilmot, Middleport
Carl G. Leo Wolf, Niagara Falls
E. W. Wollaber, Cambria
W. C. Wood, Lockport
Number of Members, 49.

MEDICAL SOCIETY OF THE COUNTY OF ONEIDA.

MEETINGS.—Annual, second Tuesday in April; semi-annual, second Tuesday in October; quarterly, second Tuesday in July and January. The semi-annual meeting is held in Rome; the others in Utica.

Officers. (April, 1903.)

Charles Bernstein, *President.* G. J. Pollard, *Vice-President.*
L. F. Pattengill, *Secretary.* Herbert G. Jones, *Treasurer.*
Walter Gibson, *Librarian.*

Censors: W. C. Gibson, H. L. Borland, F. H. Peck, B. P. Allen.

Members.

(Where no town is mentioned Utica is to be understood.)

E. P. Abbott, Oriskany
B. P. Allen, Oriskany
James A. Armstrong, Clinton
Raymond L. Baker, Clarendon Bldg
Smith Baker, Winston Building
D. A. Barnum, Cassville
Charles Bernstein, Rome
Thomas J. Bergen, 93 John st
H. L. Borland, Camden
H. C. Bradford, Westernville
F. H. Brewer, Utica
A. J. Browne, Carlisle Building
Henry E. Brown, Utica
W. H. Brownell, Utica
Wales Buell, Walesville
P. J. Campbell, Utica
E. P. Clark, Utica
Wallace Clarke, 136 Park av
A. M. Collier, Utica
Ira M. Comstock, New York Mills
Mathias Cooke, 92 Columbia st
Frank D. Crim, Utica
C. W. Crumb, Utica
Harriet Cutter, Utica
Morris J. Davies, Utica
Theodore Deecke, Utica
F. B. Delaney
Albert Dietrich, Rome
A. P. Dodge, Oneida Castle
C. V. J. Doolittle, Utica
James W. Douglass, Boonville
Frederick J. Douglass, Utica
H. G. Dubois, Camden
Roy B. Dudley, Deansboro
D. C. Dye, Carlisle Building
Eliza M. Ellinwood, Rome
E. P. English, Boonville
Thomas H. Farrell, Utica
G. M. Fisher, Clayville
John F. Fitzgerald, Rome
W. E. Ford, 266 Genesee st
Conway Frost, Rome
Earl D. Fuller, 148 Columbia st
F. A. Gary, Vernon
W. C. Gibson, State Hospital, Utica
William M. Gibson, 260 Genesee st
S. L. Gifford, Whitesboro
A. A. Gillette, Rome
J. H. Glass, 246 Genesee st
Giovanni Grana, Utica
V. B. Hamlin, Clinton
Joseph V. Haberer, 66 Miller st
C. R. Hart, New Hartford
A. L. Holden, 132 South st
Ira D. Hopkins, 25 Bladina st
Howard F. Hubbard, Rome
J. G. Hunt, 192 Genesee st
Myron W. Hunt, Holland Patent
E. M. Hyland, 221 Genesee st
Arthur M. Johnston, Utica
Herbert G. Jones, Utica
J. D. Jones, Utica
James E. Jones, Clayville
Leroy H. Jones, Utica
Thomas Z. Jones, Waterville
William W. Jones, Rome
Judson G. Kilbourn, 247 Genesee st
D. R. Kinlock, Utica
George A. Krug, Utica
William Kuhn, Rome
R. A. Lees, Utica
G. N. Lehr, Rome

G. M. Lewis, Vernon
James McCullough, Remsen
W. R. Marsden, Utica
Angeline Martine, Utica
S. C. Maxon, Utica
Julia E. Merchant, Rome
James Middleditch, Rome
Frederick Miller, Deerfield
Wm. Moffat, Utica
W. S. Morris, Utica
Charles Munger, Knoxboro
Stephen H. Murphy, Utica
William H. Nelson, Taberg
H. Nicholas, North Bay
Thomas G. Nock, Jr., Rome
H. C. Palmer, 253 Genesee st
H. L. Palmer, Utica State Hospital
W. B. Palmer, 30 South st
L. F Pattengill, 225 Genesee st
F. H. Peck, Utica
W. D. Peckham, Utica
Johnson Pillmore, Delta
G. G. J. Pollard, Oriskany Falls
William Powell, 351 Genesee st
H. Frank Preston, Utica
Medina Preston, Sangerfield
W. K. Quackenbush, Trenton
Hamilton S. Quin, 238 Genesee st
William B. Reid, Rome
Lafayette Rinkle, Booneville
D. H. Roberts Utica
W. B. Roemer, Utica
J. M. Ross, Utica
Charles P. Russell, 196 Genesee st
W. D. Russell, New Hartford
W. J. Schuyler, 266 Genesee st
Thos. P. Scully, Rome
George Seymour, 227 Genesee st
Charles W. Shaver, Florence
A. R. Simmons, Utica
Hugh Sloan, 142 Columbia st
A. H. Smith, Camden
Frederick W. Smith, Utica
Charles E. Smith, Whitesboro
Edward J. Stevens, 183 Genesee st
Henry C. Sutton, Rome
J. A. Stranahan, Rome
L. Swartwout, Prospect
J Nelson Teeter, Utica
Charles B Tefft, Utica
H. J Teller, Rome
William M. Townsley, Camden
Charles G. Ward, Utica
Charles R. Weed, Utica
W. E. Wetmore, Utica
Henry D. White, Rome
J. F. Wingenback, Utica

Number of Members, 128.

Deceased: William R. Davis, Utica, April 19, 1903, æt. 47; Francis T. Gorton, Waterville, July 28, 1902, æt. 58.

MEDICAL SOCIETY OF THE COUNTY OF ONONDAGA.

(Organized in 1806.)

MEETINGS.—Annual, second Tuesday in May; semi-annual, third Tuesday in December; quarterly, at such intermediate times as the Society may direct. All meetings are held at Syracuse.

Officers. (May, 1903.)

E. J. Wynkoop, *President.*
A. S. Hotaling, *Secretary.*
J. H. Burch, *Vice-President.*
F. H. Flaherty, *Treasurer.*

Members.

(Where the town is not mentioned Syracuse is to be understood.)

Henry B. Allen, Baldwinsville
W. J. Ayling, 606 E. Fayette st
Archer D. Babcock, Fayette Park
Theresa Bannan, 503 Warren st
H. C. Baum, University Block
Charles H. Benson, 234 Seymour st
O. A. Blumenthal, 611 Warren st
A. B. Breese, James st
George B. Broad, 742 Cortland av
G. L. Brown, Euclid
J. W. Brown, Mottville
George J Bryan, Fayetteville
J H. Burch, Baldwinsville
Robert Burns, 806 S. Salina st
C. Floyd Burrows, 824 E. Genesee st
J. J. Buettner, 1004 S. Salina st
James E. Carr, Jordan
J. C. Carson, State Asylum
B. F. Chase, East Syracuse
George E. Clark, Skaneateles
Gaylord P. Clark, W. Genesee st
Frank R. Coe, Warners
J. H. Coe, 804 E. Genesee st
Allen Cone, Elbridge
George W. Cook, 208 W. Fayette st
C. E. Coon, 509 S. Salina st
S. Ellis Crane, Onondaga Valley
S Boyce Crayton, University Block
M. E. Cregg, South Onondaga
M. H. Cullings, 321 James st
William A Curtin, Fayette Park
M. A. Curtiss, Kirkville

A. J. Dallas, E. Fayette st
Chas. N. Daman, 621 E. Genesee st
O. G. Dibble, Pompey
H. D. Didama, 424 S. Salina st
F. O. Donohue, Warren st
A. G. Doust, 302 Delaware st
F. E. Easton, 1105 E. Genesee st
A. S. Edwards, 1506 N. Salina st
G. A. Edwards, S. Salina st
H. L. Elsner, Fayette Park
Du Grand B. Erway, East Syracuse
J. T. D. Fischer, N. Salina st
M. B. Fairchild, 1305 N. Salina st
C. A. Felton, W. Genesee st
Frederick Flaherty, Warren st
C. H. Graves, Delphi
M. E. Gregg, South Onondaga
W. A. Groat, E. Onondaga st
T. H. Halsted, University Block
R. C. Hanchett, Warren st
Hiram B. Hawley, 1100 N. Salina st
A. D. Head, 202 W. Genesee st
C. E. Heaton, Baldwinsville
A. W. Hedden, S. Salina st
J. L. Heffron, 448 S. Salina st
Mark Heiman, 320 Montgomery st
A. S. Hotaling, 801 E. Genesee st
Nathan Jacobson, 430 S. Salina st
J. Robert Johnson, Warren st
Edward B. Kaple, Elbridge
W. C. Kellogg, 203 Seymour st
J. W. Knapp, 432 W. Genesee st
A. E. Larkin, 334 Montgomery st
William F. Law, 602 Lodi st
T. Harris Levy, E. Genesee st
G. Griffin Lewis, University Block
Hersey G. Locke, 507 E. Genesee st
B. W. Loomis, 1033 S. Salina st
B. C. Loveland, Fayette Park
M. M. Lucid, Tully
Charles E. McCrary, 110 South av
H. A. MacGruer, University Block
W. C. McKeeby, 312 S. Warren st
D. J. McLaughlin, Skaneateles Falls
R. C. McLennan, 515 S. Salina st
P. R. McMaster, S. Salina st
Frank McMorrow, 508 James st
F. W. Marlow, University Block
A. W. Marsh, E. Syracuse st
Edwin R. Maxson, 818 Madison st
E. S. Maxson, 818 Madison st
Wm. H. May, 800 E. Fayette st
Alfred Mercer, 324 Montgomery st
A. C. Mercer, 324 Montgomery st
E. B. Merwin, Manlius
H. D Merwin, Cicero
A. B. Miller, 326 Montgomery st
E. L. Mooney, 416 Warren st
F. L. Morgan, E. Fayette st
N. A. Munroe, 424 James st
W. W. Munson, Otisco
D. H. Murray, University Block
O. W. Oberlander, 310 Ash st
Charles L. Parker, Onondaga
Israel Parsons, Marcellus
George M. Price, Warren st
A. B. Randall, S. Salina st
C. S. Roberts, 212 W. Willow st
Arthur H. Rood, Minoa
M. R. Rood, 422 Warren st
Joseph C. Roth, 435 Gifford st
A. S. Ruland, 300 Delaware st
W. G. Russell, Watkins Glen, N. Y.
F. S. Sampson, North Syracuse
L. A. Saxer, 305 James st
F. W. Sears, 709 S. Warren st
John C. Shroudy, 1706 W. Genesee st
I. M. Slingerland, Fayetteville
F. W. Slocum, Camillus
F. W. Smith, S. Salina st
S. F. Snow, 707 University Block
H. L. Stebbins, 125 E. Onondaga st
F. H. Stevenson, 307 Warren st
F. A. Strong, Brewerton
P. C. Ten Eyck, Montgomery st
D. M. Totman, 303 Montgomery st
A. F Vadeboncœur, James st
Eli Van de Warker, 404 Genesee st
E. S. Van Duyn, 318 James st
John Van Duyn, 318 James st
Charles J. Walch, 1221 S. Salina st
William L. Wallace, Fayette Park
H. A. Weed, Jordan
Charles E. Weidman, Marcellus
W. J. Werfleman, 908 N. Salina st
R. A. Whitney, Liverpool
Hiram Wiggins, Elbridge
N. Wilbur, Fayetteville
H. B. Wright, Skaneateles
J. S. Wright, Plainville
E. J. Wynkoop, 406 James st

Number of Members, 133.

Deceased: Margaret Stanton, Syracuse, Jan. 22, 1903, æt. 58; N. H. Curtis, Manlius, March, 1903, æt. 54.

MEDICAL SOCIETY OF THE COUNTY OF ONTARIO.

MEETINGS.—Annual, second Tuesday in July; quarterly, second Tuesday in October, January, and April.

Officers.

C. C. Thayer, *President.* J. H. Jewett, *Vice-President.*
D. A. Eiseline, *Secretary and Treasurer.*

Members.

Herman J. Abel, Honeoye
A. D. Allen, Gorham
D. S. Allen, Hall's Corners
Herbert E. Baright, Clifton Springs
A. L. Beahan, Canandaigua
H. C. Buell, Canandaigua
D. R. Burrell, Canandaigua
M. R. Carson, Canandaigua
Henry D. Clapp, Geneva
William B. Clapper, Victor
James J. Collie, Geneva
J. Pope Le Laney, Geneva
J. F. Draper, Victor
H. M. Eddy, Geneva
Daniel A. Eiseline, Shortsville
O. J. Hallenbeck, Canandaigua
W. S. Hicks, Bristol
W. A. Howe, Phelps
John Hutchins, Cheshire
Mary L. Jennings, Geneva
J. H. Jewett, Canandaigua
H. J. Knickerbocker, Geneva
B. O. Kinnear, Clifton Springs
C. C. Lytle, Geneva
C. D. McCarthy, Geneva
F. E. McClellan, Canandaigua
George McClellan, Canandaigua
B. T. McDowell, Bristol Centre
D. J. Mallery, Bristol Centre
W. McCaw, Geneva
Wm. Austin Macy, Willard
O. V. Mason, Farmington
A. M. Mead, Victor
C. P. W. Merritt, Clifton Springs
Charles F. Nieder, Geneva
W. F. Powers, Stanley
J. R. Pratt, Manchester
John H. Pratt, Manchester
John A. Robson, Hall's Corners
T. D. Rupert, Geneva
J. A. Sanders, Clifton Springs
George W. Sargent, Seneca Castle
E. B. Sayre, Allen's Hills
W. W. Skinner, Geneva
F. W. Spalding, Clifton Springs
John A. Spengler, Geneva
F. Lansing Stebbins, Geneva
Geo. E. P. Stevenson, Gorham
C. C. Thayer, Clifton Springs
William Turck, Clifton Springs
F. D. Vanderhoof, Phelps
George E. Walker, Geneva
S. R. Wheeler, E. Bloomfield
L. F. Wilbur, Honeoye
G. B. Young, Geneva

Number of Members, 55.

MEDICAL SOCIETY OF THE COUNTY OF ORANGE.

(Organized July 1, 1806.)

MEETINGS.—Annual, first Tuesday in May; semi-annual, first Tuesday in October.

Officers. (May, 1903.)

C. E. Townsend, *President.* C. I. Redfield, *Vice-President.*
M. A. Stivers, *Secretary.* W. I. Purdy, *Treasurer.*
Censors: Chas. N. Skinner, W. T. Seeley, W. J. Carr, G. F. Pitts, I. H. Lent.
Committee on Hygiene: F. W. Dennis, E. R. Elliott, D. T. Condict.

Members.

A. E. Adams, Newburgh
A. Herbert Bayard, Cornwall
William Beattie, Cornwall
George S. Bond, Washingtonville
H. K. Bradner, Warwick
W. B. Bradner, Warwick
E. F. Brooks, Newburgh
G. Herbert Brown, Highland Falls
J. D. Brownell, Walden
C. W. Butler, Cornwall
W. H. Burke, Middletown
William J. Carr, Newburgh
D. T. Condict, Goshen
Clinton G. Cooley, Montgomery
James C. Coleman, Goshen
William L. Cuddeback, Port Jervis
J. O. Davis, Howells
C. W. Dennis, Goshen
F. W. Dennis, Unionville
John Deyo, Newburgh
L. G. Distler, Westtown
William E. Douglas, Middletown
Matthew H. Dubois, Washingtonville
Charles Duffy, Newburgh
Mary Ella Dunning, Newburgh
William L. Dunning, Newburgh
E. Ross Elliott, Montgomery
G. A. Emory, Middletown
William Evans, Westtown
A. P. Farries, Florida
Frank W. Gerecke, Newburgh
L. L. Gillette, Turners
W. S. Gleason, Newburgh
Thomas S. Gilson, Middletown

A. M. Goodman, Jr., Cornwall
Charles H. Hall, Monroe
Lewis Hanmore, Newburgh
Daniel B Hardenbergh, Middletown
H. Hardenbergh, Port Jervis
William Hollinger, Newburgh
John T. Howell, Newburgh
Joseph B. Hulett, Middletown
C. N. Knapp, Port Jervis
Isaac H. Lent, Middletown
A. V. Jova, Newburgh
D. G. Lippincott, Campbell Hall
Raphael Medrick, Port Jervis
T. D. Mills, Middletown
L. J. Merritt, Pine Bush
F. D. Myers, Slate Hill
E. A. Nugent, Unionville
Clarence Ormsbee, Newburgh
J. B. Peters, Walden
L. R. Pierce, Newburgh
G. Fred. Pitts, Warwick
Edgar Potts, Port Jervis
Albert W. Preston, Middletown
Willis I. Purdy, Middletown
Charles I. Redfield, Middletown
H. H. Robinson, Goshen
A. Santee, Scotchtown
W. T. Seeley, Amity
Charles N. Skinner, Port Jervis
D. B. Smiley, Middletown
C. P. Smith, Jr., Chester
David H. Sprague, Central Valley
Moses A. Stivers, Middletown
Henry B. Swartwout, Port Jervis
Robert Taylor, Otisville
Charles E. Townsend, Newburgh
J. H. Thompson, Goshen
H. A. Waldron, Newburgh
W. W. Wendover, Warwick
Edward Dubois, Woodhall, Monroe
Warren Worcester, Middletown
Theodore Writer, Otisville
Number of Members, 77.

Deceased: S. G. Carpenter, Chester, Sept. 13, 1901, æt. 76; Joseph Moffat, Washingtonville, Aug. 16, 1901, æt. 68.

MEDICAL SOCIETY OF THE COUNTY OF ORLEANS.

(Organized January 8, 1873.)

MEETINGS.—Annual, second Wednesday in May; second meeting, second Wednesday in September; third meeting, second Wednesday in January.

Officers. (May, 1903.)

J. J. Simonds, *President.* Mrs. E. W. Squier, *Vice-President.*
John Dugan, *Secretary and Treasurer.*

Censors: J. H. Taylor, S. R. Cochrane, McLean Caverly, H. L. James, R. W. Bamber.

Members.

E. R. Armstrong, Holley
R. W. Bamber, Kendall
M. L. Caverly, Albion
J. G. Dolley, Albion
John Dugan, Albion
W. F. Eaman, Gaines
Arthur I. Eccleston, Waterport
J. J. Eckerson, Shelby
Charles E. Fairman, Lydonville
W. R. Fitch, Knowlesville
A. R. Holcomb, Carlton
John T. James, Medina
F. L. June, Waterport
F. H. Lattin, Gaines
J. Maynard, Medina
Edward Munson, Medina
F. L. Sherwood, Albion
J. J. Simonds, Barre Centre
Mrs. E. W. Squier, Albion
J. T. Stokes, Medina
F. B. Storer, Holley
John E. Sutton, Albion
John Taylor, Holley
J. Thornton, Eagle Harbor
E. M. Tompkins, Knowlesville
Jessie Waldo, Hulberton
Mrs. Harriet N. Watson, Albion
Number of Members, 27.

Deceased: Harvey L. James, Shelby, March 14, 1903, æt. 46.

MEDICAL SOCIETY OF THE COUNTY OF OSWEGO.

(Organized in 1821.)

MEETINGS.—Annual, second Tuesday in May; semi-annual, second Tuesday in November, at Oswego.

Officers. (May, 1903.)

Charles E. Low, *President.* J. T. Dwyer, *Vice-President.*
Wm. C. Todt, *Secretary.* P. M. Dowd, *Treasurer.*
Censors: W. M. Wells, J. S. Howard, Ira L. Jones.

Members.

J. R. Allen, Orwell
W. G. Babcock, Cleveland
C. G. Bacon, Fulton
C. J. Bacon, Fulton
Howard G. Bartlett, Williamstown
Strong M. Bennett, Mexico
G. W. Betts, Pulaski
E. H. Boyd, Hannibal
W. J. Bulger, Oswego
J. L. Bulkley, Sandy Creek
H. W. Caldwell, Pulaski
C. F. Chiler, Fulton
Newton Cook, Sandy Creek
F. L. Cooley, Hannibal Centre
E. W. Crespell, Williamstown
R. F. Crockett, Sandy Creek
S. J. Crockett, Sandy Creek
E. J. Cusack, Fulton
Byron Dewitt, Oswego
R. J. Dimon, Hastings
Harriet M. Doane, Fulton
Pascal M. Dowd, Oswego
E. J. Drury, Phœnix
J. F. T. DuBois, Oswego
J. T. Dwyer, Oswego
Joseph W. Eddy, Oswego
Frank B. Foote, Parish
T. J. Green, Mexico
Albert L. Hall, Fulton
John E. Hamill Phœnix
Freeman J. Hoffman, Fulton
Leroy F. Hollis, Lacona
James S. Howard, Oswego
J. W. Huntington, Mexico
Louis Fowler Joy, Fulton
George P. Johnson, Mexico
Ira L. Jones, Minetto
W H. Kidder, Oswego
C. R. Lee, Fulton
Charles E. Low, Pulaski
F. S. Low, Pulaski
C. Macfarlane, Oswego
James E. Mansfield, Oswego
H. P. Marsh, Fulton
Eva E. McKnight, Oswego
J. L. More, Pulaski
D. D. O'Brien, Oswego
G. W. Nelson, Orwell
Melvin Ernest Paige, Oswego
Joseph Pero, West Amboy
H. D. C. Phelps, Oswego
S. A. Russell, Fulton
William O. Scott, Parish
Charles A. Sheridan, Oswego
G. Franklin Smith, Mexico
James K. Stockwell, Oswego
William C. Todt, Oswego
Melzar B. Veeder, Central Square
W. M. Wells, Fulton
G. G. Whitaker, Fulton
Hadwen P. Wilcox, Central Square
R. A. Wilcox, Phœnix

Number of Members, 61.

Deceased: R. N. Cooley, Hannibal Centre, July 15, 1901, æt. 69; C. M. Lee, Fulton, November 28, 1901, æt. 77; Elvira Ranier, Oswego, October 12, 1902, æt. 54; Lucien H. Shepherd, Oswego, January 18, 1903, æt. 31.

MEDICAL SOCIETY OF THE COUNTY OF OTSEGO.

(Organized in 1806.)

MEETINGS.—Annual, fourth Tuesday in May, at Cooperstown; semi-annual, third Tuesday in January.

Officers. (May, 1903.)

B. W. Dewar, *President.* Rupert W. Ford, *Vice-President.*
H. W. Boorn, *Secretary.* James Burton, *Treasurer.*
Censors: H. D. Sill, C. E. Parish, J. H. Moon.

Members.

C. S. Arnold, Mt. Vision
M. Imogene Bassett, Cooperstown
Wilson T. Bassett, Cooperstown
B. F. Bishop, Garrattsville
A. D. Blakely, Milford
H. D. Blanchard, Portlandville
Henry W. Boorn, Schenevus
A. H. Brownell, Oneonta
James Burton, Cooperstown
Andrew J. Butler, Unadilla

William B. Campbell, Edmeston
Meigs Case, Oneonta
Lorenzo B. Chapman, Oneonta
Charles H. Chesbro, Unadilla Forks
H. H. Clapsaddle, Toddsville
Harry L. Cruttenden, Cooperstown
A. W. Cutler, Oneonta
David H. Davis, East Worcester
B. W. Dewar, Cooperstown
M. K. Engell, South Valley
Rupert W. Ford, Otego
John W. Green, West Laurens
E. E. Houghton, Schenevus
F. H. Hurst, Oneonta
Marshal Latcher, Oneonta
Horace Lathrop, Cooperstown
W. R. Lough, Edmeston Centre
J. M. McClellan, Oneonta
D. B. Manchester, Oneonta
J. H. Martin, Binghamton
George L. Merritt, Cherry Valley
John H. Moon, Cooperstown
G. S. Olin, Oneonta
C. E. Parish, Maryland
Edward J Parish, New York
Ozias W. Peck, Oneonta
S. G. Pomeroy, West Oneonta
George E. Schoolcraft, Hartwick
W. R. Seeber, Milford
Henry D. Sill, Cooperstown
George A. Sloan, Westford
Julian C. Smith, Oneonta
J. W. Sterricker, Roseboom
J. W. Swanson, Springfield Centre
Frank L. Windsor, Laurens
Milton C. Wright, Mt. Vision

Number of Members, 46.

Deceased: B. A. Church, Oneonta, June 5, 1902, æt. 47; J. K. Leaning, Cooperstown, April, 1902; J. Kerney Strong, Laurens, April, 1903; G. C. Thayer, Cooperstown, April, 1903.

MEDICAL SOCIETY OF THE COUNTY OF PUTNAM.

(Organized July 29, 1879.)

MEETINGS.—Annual, fourth Tuesday in April; semi-annual, fourth Tuesday in October.

(Society at present not in active existence.)

MEDICAL SOCIETY OF QUEENS-NASSAU.

MEETINGS.—Annual, last Tuesday in May; semi-annual, last Tuesday in October.

Officers. (May, 1903.)

Henri M. Auger, *President.* Frank T. de Lano, *Vice-President.*
James S. Cooley, *Secretary and Treasurer.*
John Ordronaux, *Historian.*

Members.

A. J. Anderson, Long Island City
H. M. Auger, Jamaica
H. L. Barker, Woodside
Irving F. Barnes, Oyster Bay
J. H. Barry, Long Island City
Austin J. Blanchard, Jamaica
J. H. Bogart, Roslyn
Samuel G. Bradbury, Lynbrook
Willet S. Brown, Flushing
P. H. Bumster, Long Island City
W. J. Burnett, Long Island City
Barton W. Brush, Woodside
James E. Burns, Glen Cove
Martin F. Burns, Long Island City
J. F. Bloodgood, Flushing
M. S. Caldwell, Far Rockaway
Edwin Carman, Freeport
G. Herbert Carter, Huntington
William I. Cocke, Port Washington
A. C. Combes, Newton
James S. Cooley, Glen Cove
G. W. Donahue, Northport
Horace D. Dow, Maspeth
F. T. de Lano, Rockville Centre
John W. Durkee, Sea Cliff
G. D. Farwell, Elmhurst
George W. Faller, Oyster Bay
C. G. J. Finn, Hempstead
Neil O. Fitch, Astoria
Walter G. Frey, Long Island City
G. A. Fensterer, Floral Park
George Forbes, L. I. City
J. Mansfield Foster, Valley Streams
William B. Gibson, Huntington
E. A. Goodridge, Flushing
R. C. Gray, Jamaica
George H. Hammond, Freeport
Samuel Hendrickson, Jamaica
Skidmore Hendrickson, Brooklyn
M. W. Herriman, Long Island City

J. P. Heyen, Northport
J. R. Hinkson, Long Island City
J. E. Hutcheson, Rockville Centre
Arthur D. Jaques, Lynbrook
James B. Kennedy, Long Island City
J. J. Kindred, Astoria
L. N. Lanehart, Hempstead
T. A. Lehmann, Long Island City
Water Lindsay, Huntington
C. H. Ludlam, Hempstead
Wm. J. Malcolm, Jericho
Henry McDonald, Morris Park
R. F. Macfarlane, Long Island City
John Mann, Westbury
P. J. McKeown, Long Island City
Paul O. Meyer, Long Island City
G. K. Meynen, Jamaica
James T. Michel, Farmingdale
J. P. Moore, Astoria
W. H. Namack, Far Rockaway
James L. New, Long Island City
C. M. Niesley, Manhasset
John Ordronaux, Roslyn
E. N. Pershing, Lawrence
Adolph G. Rave, New Hyde Park
E. G. Rave, Hicksville
R. F. B. Seaman, Locust Valley
E. D. Skinner, Mineola
W. T. Scovil, Richmond Hill
M. M. Slocum, Freeport
S. B. Smallwood, Astoria
Wm. J. Steele, Baldwin
C. B. Story, Bayside
Benj. G. Strong, Long Island City
J. D. Trask, New York City
J. C. Vanderveer, Mineola
Anselm A. Voegtle, Long Island City
Herman G. Wahlig, Sea Cliff
H. M. Warner, Hempstead
Benjamin Wood, Flushing
Philip M. Wood, Jamaica
William H. Zabriskie, Glen Cove

Number of Members, 82.

Deceased: Obed L. Lusk, Jamaica, June 20, 1902; Herbert B. Tingley, Oceanus, January 14, 1903, æt. 36; John F. Valentine, Richmond Hill, February 5, 1903, æt. 45.

MEDICAL SOCIETY OF THE COUNTY OF RENSSELAER.

(Organized 1806.)

MEETINGS.—Annual, second Tuesday in May; stated, second Tuesday in each month, except July and August.

Officers. (May, 1903.)

William Finder, Jr., *President.* M. Keenan, *Vice-President.*
William Kirk, Jr., *Secretary.* Reed Brockway Bontecou, *Treasurer.*
Censors: J. C. Hutchinson, R. A. Kirkpatrick, James Love.

Members.

(Where no town is mentioned Troy is to be understood.)

A. F. Adt, 54 4th st
W. Aikin, 12 4th st
C. S. Allen, Greenbush
W. L. Allen, Greenbush
Joseph E. Baynes, 2419 5th av
Edward W. Becker, 30 Fourth st
J. H. Bissell, 2187 5th av
R. B. Bontecou, 82 4th st
R. Brinsmade Bontecou, 82 4th st
B. S. Booth, 21 1st st
G. A. Bradbury, 543 2d av N.
E. A. Brown, 4 Union pl
Archibald Buchanan, 128 3d st
Donald G. Buchanan, 1930 5th av
E. W. Capron, 518 2d av N.
J. H. Cipperly, 131 2d st
Andrew C. Crouse, Melrose
A. D. Davidow, 141 3d st
H. E. DeFreest, 745 River st
M. D. Dickinson, 6 St. Paul's pl
T. G. Dickson, 1829 5th av
Hiram Elliott, Marshall Infirmary
M. Felter, 1626 5th av
William Finder, Jr., 2 Union pl
W. J. Fleming, 169 3d st
H. C. Gordinier, 89 4th st
C. E. Greenman, 104 3d st
T. H. Hannan, Hoosick Falls
J. B. Harvie, 1741 5th av
T. B. Heimstreet, 14 Division st
C. B. Herrick, 40 State st
W. L. Hogeboom, 2179 5th av
D. W. Houston, 18 2d st
J. C. Hutchinson, 328 Congress st
H. T. Ives, 8 15th st
M. Keenan, 2275 5th av
O. F. Kinloch, 16 4th st
Rollin A. Kirkpatrick, 739 River st
William Kirk, 2655 Fifth av
James Love, 9 Winslow av
E. L. Lyons, 298 4th av
J. P. Marsh, 1828 5th av

W. P. Mason, Prof. Chem., R. P. I.
G. L. Meredith, 1926 5th av
J. McChesney, Pawlings av
James T. McKenna, 112 4th st
E. W. Morehouse, 99 2d st
J. W. Morris, 1823 5th av
C. E. Nichols, 25 1st st
M. F. Phelan, 131 3d st
E. E. Richards, Averill Park
Z. Rosseau, 17 2d st
T. B. St. John, Centre Brunswick
W. W. Seymour, 105 3d st
S. A. Skinner, Hoosick Falls
Frederick A. Smith, 3 Clinton pl
Robert Thomson, 1643 5th av
C. Howard Travell, 1915 5th av
L. G. Tuttle, 73 4th st
B. J. Ward, 1 Cyprus st
R. H. Ward, 53 4th st
M. A. Wheeler, 763 Pawling av
F Winship, Eagle Mills
Isaac N. Wright, Averill Park
Thomas G. Wright, 17 River st
Number of Members, 65.

MEDICAL SOCIETY OF THE COUNTY OF RICHMOND.

MEETINGS.—Annual, second Wednesday in January; stated, second Wednesday in each month at the Staten Island Academy, New Brighton.

Officers. (January, 1903.)

William Bryan, *President.* George P. Jessup, *Vice-President.*
H. W. Patterson, *Secretary and Treasurer.*

Censors: Henry C. Johnston, Jefferson Scales, Alfred H. Thomas.

Members.

Preston B. Baillache, Clifton
Edwin C. Baldwin, Rosebank
F. T. Barber, Port Richmond
H. Beyer, Stapleton
Horace B. Blan, Port Richmond
W. W. Bostwick, Sailors' Snug Harbor
George P. Boulden, Tompkinsville
W. T. Bourne, Tomkinsville
William Bryan, W. New Brighton
E. J. Callahan, W. New Brighton
J. S. Carman, Castleton Corners
N. D. Chapman, Linoleumville
F. E. Clarke, W. New Brighton
James G. Clarke, W. New Brighton
E. D. Coonley, Port Richmond
Frederick Coonley, Port Richmond
L. M. Cornelius, Port Richmond
A. D. Decker, Prince's Bay
F. De Revere, Stapleton
J. L. Devlin, New Dorp
J. D. Dickson, New Brighton
A. H. Doty, Rosebank
R. Henry Golder, Rossville
H. T. Goodwin, Stapleton
J. B. Greene, Clifton
Ernest E. Hillyer, Port Richmond
G. P. Jessup, New Dorp
H. C. Johnston, New Brighton
H. C. Joy, New Brighton
Max Krueger, Stapleton
J. J. O'Dea, Stapleton
Dorothea Orr, W. New Brighton
F. Mechtold, Stapleton
Isaac L. Millspaugh, Richmond
George Mord, Stapleton
H. W. Patterson, Tompkinsville
Charles E. Pearson, Tompkinsville
Jefferson Scales, Tompkinsville
A. C. Smith, Clifton
E. H. Sparks, Prince's Bay
J. T. Sprague, Stapleton
A. H. Thomas, Clifton
C. W. Townsend, New Brighton
Wm. T. Van Vredenburgh, New Brighton
J. E. Vidal, Stapleton
J. E. Walker, Rosebank
W. C. Walser, W. New Brighton
Walker Washington, Tottenville
S. E. Whitman, Port Richmond
W. B. Wilkinson, New Brighton
E. D. Wisely, Port Richmond
Alfred S. Wood, New Brighton
J. W. Wood, Port Richmond
Number of Members, 54.

Deceased: Theodore Walser, New Brighton, April 23, 1902.

MEDICAL SOCIETY OF THE COUNTY OF ROCKLAND.

(Reorganized May 18, 1850.)

MEETINGS.—Annual, last Tuesday in May; semi-annual, last Tuesday in October; stated, last Tuesday in January and August.

Officers.

S. Demarest, *President.* J. Sengstacken, *Vice-President.*
F. E. Paget, *Secretary.* J. M. Hasbrouck, *Treasurer.*
Censors: S. Demarest, J. Sengstacken, F. E. Paget.

Members.

S. Demarest, Suffern
B. V. Dolph, Suffern
John Gillette, Sloatsburgh
L. L. Gillette, Suffern
J. M. Hasbrouck, Garnerville
E. B. Laird, Haverstraw
F. E. Paget, Spring Valley
J. Sengstacken, Stony Point
W. S. Taylor, Spring Valley
N. B. Van Houten, New City
T. C. Wood, Haverstraw
Number of Members, 11.

MEDICAL SOCIETY OF THE COUNTY OF ST. LAWRENCE.
(Reorganized 1861.)

MEETINGS.—Annual, first Tuesday in April; semi-annual, first Tuesday in October.

Officers. (1903.)

James Wiltse, *President.* E. M. Somers, *Vice-President.*
S. W. Close, *Secretary.* A. H. Allen, *Treasurer.*
Censors: Jesse Reynolds, B. F. Drury, E. H. Bridges.

Members.

Andrew H. Allen, Gouverneur
F. D. Allen, Richville
W. L. Babcock, Ogdensburg
L. B. Baker, Russell
John N. Basset, Canton
E. H. Bridges, Ogdensburg
S. E. Brown, Ogdensburg
S. Pope Brown, Potsdam
C. M. Burdick, Ogdensburg
S. W. Close, Gouverneur
W. Grant Cooper, Ogdensburg
F. W. Dalmage, Hermon
W. S. Daly, Ogdensburg
S. W. Dodge, Massena
J. MacB. Davidson, Potsdam
Benjamin F. Drury, Gouverneur
Fred. F. Drury, Gouverneur
Franklin D. Earl, Ogdensburg
J. Q. Flood, Hopkinton
Frank J. Fuller, Potsdam
E. H. Hackett, North Lawrence
W. H. Hanbidge, Ogdensburg
C. B. Hawley, Gouverneur
Lucia E. Heaton, Canton
M. D. Hooks, Madrid
R. H. Hutchings, Ogdensburg
H. Kay Kerr, Hammond
D. Kellock, Fullerville
W. H. Kidder, Ogdensburg
Roy L. Leak, Ogdensburg
William Mabon, New York City
Grant C. Madill, Ogdensburg
Alex. S. McEwen, Ogdensburg
O. McFadden, Massena
H. A. McIlmoyl, Ogdensburg
E. A. Markley, Edwards
E. A. Nevin, Brasher
N. A. Pashayan, Ogdensburg
Caroline Pease, Ogdensburg
T. A. Pease, Norwood
Chas. Pomeroy, Ogdensburg
C. A. Rutherford, Sprague ville
Jessie Reynolds, Potsdam
G. M. Russell, Massena
P. H. Shea, Canton
Moses E. Smith, Colton
W. C. Smith, Winthrop
E. M. Somers, Jr., Ogdensburg
H. S. Stilwell, Ogdensburg
D. M. Taylor, Edwards
Frank F. Taylor, Massena
S. D. Wilgus, Ogdensburg
J. C. Wilson, Canton
James Wiltse, Benson's Mines
W. D. Witherbee, Potsdam
Number of Members, 55.

MEDICAL SOCIETY OF THE COUNTY OF SARATOGA.
(Organized in July, 1807.)

MEETINGS.—Annual, first Tuesday in June; semi-annual, first Tuesday in December.

Members. (List not revised.)

MEDICAL SOCIETY OF THE COUNTY OF SCHENECTADY.

(Organized January 16, 1869.)

MEETINGS.—Annual, second Tuesday in January; semi-annual, second Tuesday in June; monthly, second Tuesday in February, March, April, May, September, November, and December.

Officers. (January, 1903.)

Edward J. Wiencke, *President.* Reed A. Sauter, *Vice-President.*
William L. Fodder, *Secretary.* G. V. Johnson, *Treasurer.*

Censors: W. L. Huggins, Peter McPartlon, W. A. Stone.

Members.

W. A. Alexander, Schenectady
Herman Bauer, Schenectady
Lester Betts, Schenectady
C. G. Briggs, Schenectady
H. A. Bryant, Mt. Pleasant
F. F. Burtis, Schenectady
Thomas Carner, Schenectady
C. F. Clowe, Schenectady
W. T. Clute, Schenectady
J. H. Collins, Schenectady
C. C. Duryea, Schenectady
A. Ennis, Pattersonville
John Fallon, Schenectady
A. S. Fay, Bellevue
Wm. L. Fodder, Schenectady
Fenton I. Gidley, Schenectady
Reed Gilmore, Schenectady
W. W. Goddard, Schenectady
W. G. B. Hall, Schenectady
Stillman S. Ham, Schenectady
J. A. Heatley, Schenectady
W. L. Huggins, Schenectady
H. Jackson, Mount Pleasant
G. V. Johnson, Schenectady
D. L. Kathan, Schenectady
Henry A. Kurth, Schenectady
U. Kingman, Scotia
M. S. Lord, Schenectady
C. A. MacMinn, Schenectady
George E. McDonald, Schenectady
Ralph McDougall, Delanson
J. F. McEncroe, Schenectady
Charles McMullen, Schenectady
Peter McPartlon, Schenectady
P. T. Markey, Schenectady
Janet Murray, Schenectady
H. V. Mynderse, Schenectady
William Parsons, Fultonville
William L. Pearson, Schenectady
L. G. Price, Schenectady
M. G. Plank, Schenectady
Joseph Raymond, Schenectady
J. E. Reed, Schenectady
E. T. Rulison, Schenectady
Reed A. Sauter, Schenectady
J. L. Schoolcraft, Schenectady
Harman A. Staley, Schenectady
Walter A. Stearns, Schenectady
W. A. Stone, Schenectady
G. B. Teames, Schenectady
C. N. Van Den Burgh, Bellevue
Frank Vander Bogert, Schenectady
E. S. Vass, Schenectady
A. Van Vranken, Schenectady
Burton Van Zandt, Schenectady
Edward J. Wiencke, Schenectady
William Wilson, Scotia
Edwin Young, West Glenville

Number of Members, 58.

MEDICAL SOCIETY OF THE COUNTY OF SCHOHARIE.

(Organized in 1806; reorganized in 1857.)

MEETINGS.—Annual, first Tuesday in June; semi-annual, second Tuesday in October.

Officers. (1903.)

O. A. Snyder, *President.* C. E. Weidman, *Vice-President.*
H. F. Kingsley, *Secretary.* LeRoy Becker, *Treasurer.*

Censors: C. K. Frazer, L. O. White, M. D. Lipes,
C. S. Best, F. L. Posson.

Committee on Hygiene: W. T. Rivenburgh, W. W. Burgett.

Members.

F. P. Beard, Cobleskill
LeRoy Becker, Cobleskill
C. S. Best, Middleburgh
E. E. Billing, Gilboa
J. R. Brown, Seward
E. E. Brayman, Livingstonville

E. O. Bruce, Hindsville
M. Bruce, Charlotteville
W. W. Burgett, Fultonham
L. Cross, Cobleskill
Charles Dickinson, Seward
C. K. Frazier, Cobleskill
Andrew J. Griffin, Carlisle
Abram L. Haines, Schoharie
John Hotaling, Gallupville
H. F. Kingsley, Schoharie
Ezra Lawyer, Cobleskill
D. M. Leonard, Broome Centre
M. D. Lipes, Howe's Cave
Edward Marsh, Sloansville
S. A. Mereness, Carlisle
J J. Miller, Cobleskill
D. Norwood, Esperance
H. L. Odell, Sharon Springs
Carolyn Oldendorf, Cobleskill
E. S. Persons, Gilboa
F. L. Posson, Schoharie
W. S. Rivenburgh, Middleburgh
R. J. Roscoe, Cobleskill
J. Rossman, Middleburgh
Edgar S. Simpkins, Middleburgh
Nathan Smith, Richmondville
O. A. Snyder, Schoharie
James M. Tibbits, West Fulton
John H. Weckel, Breakabeen
Chas. E. Weidman, Gallupville
Henry D. Wells, Middleburgh
C. F. Wharton, Summit
L. O. White, Sharon Springs
Number of Members, 39.

MEDICAL SOCIETY OF THE COUNTY OF SCHUYLER.

(Organized December 29, 1857.)

MEETINGS.—Annual, second Tuesday in January; semi-annual, second Tuesday in July.

Officers. (List not revised; no active Society.)

MEDICAL SOCIETY OF THE COUNTY OF SENECA.

MEETINGS.—Annual, second Thursday in May.

(*Officers.* (Not revised.)

John F. Crosby, *President.* Edwin R. Bishop, *Vice-President.*
D. F. Everts, *Secretary.* Frank M. Severson, *Treasurer.*

Censors: Elias Lester, George A. Bellows, E. W. Bogardus, John Denniston, L. A. Gould.

Members.

A. J. Alleman, McDougall's
F. F. Bamford, Willard
George A. Bellows, Waterloo
M. D. Blaine, Auburn
E. W. Bogardus, Poplar Ridge
Frederick E. Bowlby, Willard
Harvey E. Brown, Fayette
J. F. Carlton, Waterloo
J. S. Carman, Lodi
George W. Clark, Waterloo
J. F. Crosby, Seneca Falls
Thomas Currie, Willard
John Denniston, Ovid
Richard Dey, Romulus
Robert Doran, Willard
D. F. Everts, Romulus
John Flickinger, Trumansburg
Louis A. Gould, Farmer
James H. Haslett, Waterloo
Elias Lester, Seneca Falls
Frederick W. Lester, Seneca Falls
William A. Macy, Willard
James E. Medden, Seneca Falls
W. S. Nelson, Seneca Falls
J. O. Pangburn, Logan
George Post, Ovid
James S. Purdy, Seneca Falls
John Russell, Willard
Wm. L. Russell, Willard
Charles F. Sanborn, Willard
Frank W. Severne, Farmer
Frank M. Severson, Seneca Falls
William B. Stacy, Magee's Corners
J. H. Sternberg, Waterloo
William Steinbach, Willard
J. M. Townsend, Trumansburg
Number of Members, 35.

MEDICAL SOCIETY OF THE COUNTY OF STEUBEN.

MEETINGS.—Annual, second Tuesday in May; semi-annual, second Tuesday in October; quarterly, second Tuesday in August.

Officers. (1903.)

Edward J. Carpenter, *President.* James C. Dorr, *Vice-President.*
W. W. Smith, *Secretary and Treasurer.*

Censors: C. S. Parkhill, Ira P. Smith, W. S. Cobb,
E. J. Carpenter, H. R. Ainsworth.

Members.

Cassius M. Ackley, South Dansville
H. R. Ainsworth, Addison
P. L. Alden, Hammondsport
A. A. Aldrich, Addison
Eli Allison, Wayne
Warren L. Babcock, Bath
Robert Bell, Monterey
C. M. Brasted, Hornellsville
T. O. Burleson, Bath
Marcus C. Butler, Corning
Edward J. Carpenter, Corning
Charles A. Carr, Corning
D. J. Chittenden, Addison
George Conderman, Hornellsville
Willis S. Cobb, Corning
James C. Dorr, Wayland
J. B. Dudley, Kanonah
F. G. Esson, Corning
Lewis Fitzsimmons, South Putney
T. B. Fowler, Cohocton
Horatio Gilbert, Hornellsville
H. S. Gillett, Savona
George S. Goff, Corning
Charles O. Green, Hornellsville
H. H. Hubbell, Corning
Harvey P. Jack, Canisteo
Ambrose Kasson, Bath
J. G. Kelly, Hornellsville
Frank H. Koyle, Hornellsville
George W. Lane, Corning
R. G. Lawrence, Hammondsport
Thomas A. McNamara, Corning
George C. McNett, Bath
D. P. Matthewson, Bath
Henry A. May, Corning
John L. Miller, Corning
J. D. Mitchell, Hornellsville
Samuel Mitchell, Hornellsville
H. B. Nichols, Pultney
W. E. Palmer, Hornellsville
C. S. Parkhill, Hornellsville
Thomas H. Pawling, Bath
Frank Ward Ross, Elmira
George M. Skinner, Wayland
H. B. Smith, Corning
I. P. Smith, Bath
Lyman B. Smith, Hornellsville
W. W. Smith, Avoca
H. S. Sproul, Corning
C. B. Stevens, Wallace
Spencer H. Stuart, Cameron
Stephen M. Switzer, Bradford
V. B. Thorne, Woodhull
C. J. Tomer, Savona
H. L. Tyler, Corning
Bertis R. Wakeman, Hornellsville
James E. Walker, Hornellsville
Townsend Walker, Campbell
F. D. Williamson, Arkport
E. Winne, Haskinville
Henry J. Wynkoop, Bath

Number of Members, 61.

MEDICAL SOCIETY OF THE COUNTY OF SUFFOLK.

(Organized July 22, 1806.)

MEETINGS.—Annual, in April, at Riverhead, during court week; semi-annual, in October, on the day and at the place directed at the April meeting.

Officers. (April, 1903.)

A. C. Loper, *President.* G. A. Smith, *Vice-President.*
P. V. B. Fowler, *Secretary.* B. D. Skinner, *Treasurer.*
J. H. Benjamin, *Librarian.*

Censors: A. H. Terry, Charles E. Wells, Frank Overton.

Members.

Clarence A. Baker, Yaphank
T. Everett Benjamin, Shelter Island
John H. Benjamin, Riverhead
W. S. Bennett, Patchogue
Samuel Blume, Riverhead
L. A. S. Bodine, East Hampton
C. G. Brink, Central Islip
Thomas H. Charette, Good Ground
Robert G. Cornwall, Jamesport
Silas R. Corwith, Bridgehampton
F. C. Dildine, Port Jefferson
Daniel Edwards, East Hampton
L. S. Edwards, Patchogue
George T. Fanning, Smithtown Branch
P. Van Benschoten Fowler, Centre Moriches
Robert W. Fowler, Central Islip

C. F. Gardner, Westhampton Beach
H. Gates Gibson, Central Islip
Hugh Halsey, Southampton
James L. Halsey, Islip
H. E. Hewlett, Babylon
W. W. Hewlett, Babylon
M. B. Heyman, Central Islip
Frank Hinkley, Central Islip
Clarence H. Hulse, Sayville
W. A. Hulse, Bayshore
P. B. Kirchbaum, Sayville
Alice M. F. Leader, Central Islip
M. B. Lewis, East Hampton
Walter Lindsay, Huntington
A. C. Loper, Greenport
Charles A. Luce, Amityville
Frank H. Magness, Central Islip
B. F. Maney, Port Jefferson
Clarence C. Miles, Greenport
E. S. Moore, Bayshore
Edward T. Murray, Central Islip
John Nugent, Southampton
Frank Overton, Patchogue
Rose K. Owen, Central Islip
A. C. Payne, Riverhead
Edward F. Preston, Amityville
Addison Raynor, Brooklyn
B. F. Rogers, Eastport
W. H. Ross, Brentwood
M. G. Ryan, Central Islip
W. B. Savage, Central Islip
M. S. Skidmore, East Moriches
B. D. Skinner, Greenport
G. A. Smith, Central Islip
J. R. Taylor, Brooklyn
A. H. Terry, Patchogue
A. P. Van Deinse, Sayville
Irving Lee Walker, Central Islip
Charles M. Walling, Bellport
Charles E. Wells, Sag Harbor
H. H. Young, Riverhead
Number of Members, 57.

MEDICAL SOCIETY OF THE COUNTY OF SULLIVAN.

(Organized October 3, 1809.)

MEETINGS.—Annual, first Wednesday after the second Tuesday in June; semi-annual, whenever the Society directs at the annual meeting.

Officers. (List not revised; Society inactive.)

Members.

W. W. Appley, Cochecton
J. A. Cauthers, Monticello
E. Crocker, Narrowsburg
J. F. Curlette, Mountaindale
W. H. DeKay, Hurleyville
S. A. Kemp, Callicoon
G. H. Lathrop, Livingston Manor
F. A. McWilliams, Monticello
Ridley C. Paine, Bethel
Charles S. Payne, Liberty
G. F. Perry, Lackawack
Charles E. Piper, Wurtsboro
G. F. Rice, Jeffersonville
W. G. Steele, Mongaup Valley
R. C. Tuttle, Rockland
W. S. Webster, Liberty
J. L. Whitcomb, Liberty
Number of Members, 17.

MEDICAL SOCIETY OF THE COUNTY OF TIOGA.

MEETINGS.—Annual, second Tuesday in January; first quarterly, second Tuesday in April; semi-annual, second Tuesday in July; second quarterly, second Tuesday in October.

Officers. (January, 1903.)

M. B. Dean, *President.* E. E. Bauer, *Vice-President.*
A. W. Stiles, *Secretary.* J. M. Barrett, *Treasurer.*
Censors: R. D. Eastman, H. L. Knapp, S. W. Thompson,
R. S. Harnden, E. E. Bauer.

Members.

D. S. Anderson, Owego
W. L. Ayer, Owego
James M. Barrett, Owego
E. E. Bauer, Owego
G. W. Beach, Apalachin
Edward S. Beck, Owego
George M. Cady, Nichols
G. L. Carpenter, Waverly
C. W. Chidester, Newark Valley
M. B. Dean, Candor
H. T. Dunbar, Candor
R. D. Eastman, Berkshire
Walter C. Everett, Nichols
Henry A. Glover, Windham, Pa.

Officers. (May, 1903.)

Samuel Pashley, *President.* R. A. Heenan, *Vice-President.*
Henry Root, *Secretary and Treasurer.*

Censors: S. J. Banker, R. C. Davies, Lewis R. Oatman.

Committee on Hygiene: W. C. Cuthbert, J. W. Dean, William M. Rapp.

Members.

O. C. Baker, Brandon, Vt.
S. J. Banker, Fort Edward
Franklin T. Beattie, Shusan
F. H. Braymer, Knoxville, Tenn.
G. A. Bromley, Granville
Frank H. Carpenter, West Rupert
J. Cipperly, Middle Falls
James S. Cooley, Glen Cove
W. C. Cuthbert, Sandy Hill
R. C. Davies, Middle Granville
J. Wyman Dean, Sandy Hill
A. E. Falkenbury, Whitehall
C. J. Farley, Bolton
Fred. G. Fielding, Glens Falls
H. Gray, Greenwich
P. Z. Hebert, London, Eng.
Robert A. Heenan, Sandy Hill
B. R. Holcomb, Whitehall
S. B. Irwin, West Hebron
Edward Joslin, Whitehall
Charles W. Keefer, Mechanicsville
J. Knowlson, Poultney, Vt.
Charles E. Lambert, New York
J. Lambert, Salem
W. B. Madison, West Hebron
J. H. Maguire, Salem
D. M. McKenzie, Granville
Wm. B. Melick, Fort Edward
J. Millington, Greenwich
H. C. Monroe, Sandy Hill
I. T. Monroe, Granville
G. B. Murray, Greenwich
Lewis R. Oatman, Greenwich
James T. Park, Sandy Hill
Samuel Pashley, Hartford
William M. Rapp, Argyle
W. H. Renois, East St. Louis, Ill.
Henry Root, Whitehall
Sidney A. Rowe, Fort Ann
B. C. Senton, Rutland, Vt.
J. C. Sill, Argyle
George M. Stillman, Argyle
C. W. Sumner, North Granville
W. A. Tenney, Granville
T. C. Wallace, Cambridge
A. M. Young, Salem

Number of Members, 46.

MEDICAL SOCIETY OF THE COUNTY OF WAYNE.

(Organized July 2, 1823.)

MEETINGS.—Annual, second Tuesday in July, at Lyons; semi-annual, second Tuesday in January, at Newark.

Officers.

E. Hoag Lapp, *President.* G. D. Barrett, *Vice-President.*
George S. Allen, *Secretary.* Darwin Colvin, *Treasurer.*

Censors: M. A. Veeder, H. L. Chase, W. J. Coppernall, E. H. Lapp.

Members.

George Allen, Clyde
George S. Allen, Clyde
G. D. Barrett, Clyde
J. S. Brandt, Ontario
Mary A. Brownell, Newark
J. S. Campbell, Ontario
M. E. Carmer, Lyons
Robert S. Carr, Williamson
H. L. Chase, Palmyra
Darwin Colvin, Clyde
W. J. Coppernall, Newark
George A. Craft, Newark
L. B. Darling, Palmyra
Gard W. Foster, Clyde
Cyrus Fulton, Lyons
T. H. Hallett, Clyde
W. J. Hennessy, Palmyra
D. B. Horton, Red Creek
C. Jennings, Macedon
W. H. Jessup, Newark
N. E. Landon, Newark
F. A. Lewis, Walworth
E. H. Lapp, Williamson
J. F. Myers, Sodus
M. W. T. Negus, South Sodus
W. F. Nutten, Newark
James W. Putnam, Lyons
R. A. Reeves, Macedon
W. H. Richardson, Sodus
J. N. Robertson, Wolcott
H. F. Seaman, Alton
A. F. Sheldon, Lyons

L. H. Smith, Palmyra
Jennie M. Turner, Lyons
M. A. Veeder, Lyons
F. L. Wilson, Sodus Point
G. D. Winchell, Rose
George D. York, Newark
A. A. Young, Newark
Number of Members, 38.

Deceased: J. N. Arnold, Clyde, May 17, 1902, æt. 65; Timothy S. Fish, Wolcott, August 9, 1901, æt. 55.

MEDICAL SOCIETY OF THE COUNTY OF WESTCHESTER.
(Organized May 8, 1797.)

MEETINGS.—Annual, third Tuesday in May, at White Plains; stated, third Tuesday in September, November, January, and March. (Time and place of stated meetings at the option of the Society.)

Officers. (May, 1903.)

R. T. Irvine, *President.* E. I. Harrington, *Vice-President.*
D. M. Orleman, *Secretary.* S. O. Myers, *Treasurer.*
N. F. Curtis, *Curator.*

Censors: H. E. Schmidt, *Chairman,* W. S. Fleming, A. M. Campbell.

Committee on Hygiene: J. T. Gibson, *Chairman,* J. L. Porteous, C. N. Raymond.

Committee on Tuberculosis: T. F. Goodwin, *Chairman,* H. B. Brown, F. W. Shipman, E. F. Brush.

Committee on Legislation: G. C. Weiss, *Chairman,* W. D. Granger, W. F. Greene, R. T. Irvine, C. A. Knight.

Members.

S. W. Adams, Mt. Vernon
Oliver L. Austin, Tuckahoe
G. B. Balch, Yonkers
Lester C. Baldwin, Bedford
Garrett N. Banker, Yonkers
A. T. Banning, Mt. Vernon
M. W. Barnum, Ossining
P. J. Bauerberg, Yonkers
E. B. Beaudry, New Rochelle
W. A. Bell, Yonkers
A. C. Benedict, Yonkers
C. S. Benedict, 310 W. 102d st, N. Y.
James C. Bennett, Yonkers
T. P. Berens, New Rochelle
Samuel Beyea, New Rochelle
J. F. J. Blace, White Plains
T. E. Bleick, New Rochelle
J. W. Bowden, Yonkers
E. N. Brandt, Hastings-on-Hudson
H. Beattie Brown, Yonkers
Valentine Browne, Yonkers
E. F. Brush, Mt. Vernon
W. E. Bullard, Larchmont
Clarence W. Buckmaster, Yonkers
C. P. Byington, Croton
P. S. Byrne, Yonkers
P. A. Callan, Yonkers
W. M. Carhart, Peekskill
A. M. Campbell, Mt. Vernon
Elon N. Carpenter, Mamaroneck
W. J. Carpenter, Katonah
D. L. Casselmann, Purdy's Station
Charles F. Chapman, Mt. Kisco
J. F. Chapman, Katonah
George B. Clark, Armonk
E. M. Clark, Mamaroneck
E. H. Codding, New Rochelle
Philander Collard, Ossining
W. S. Coons, Yonkers
R. B. Coutant, Tarrytown
G. P. M. Curry, Mt. Kisco
Stanton Curry, Peekskill
Andrew F. Currier, Mt. Vernon
Newton F. Curtis, White Plains
F. W. Dalrymple, New Rochelle
H. G. V. De Hart, White Plains
Wm. J. Dougherty, Yonkers
Joseph W. Droogan, Westchester
E. F. Duffy, Yonkers
Carroll Dunham, Irvington
A. B. Eckerson, Mt. Vernon
W. S. Emberson, New Rochelle
Robert Condit Eddy, New Rochelle
W. S. Fleming, Mt. Vernon
Robert W. Fowler, Mt. Vernon
M. H. Foy, Yonkers
S. E. Getty, Yonkers
J. T. Gibson, Yonkers
Fred. R. Glover, Mt. Vernon
Thomas F. Goodwin, Mt. Vernon
I. T. Gorton, Yonkers
William D. Granger, Bronxville
J. P. Greene, Mamaroneck
William F. Greene, Mt. Vernon
Daniel M. Griffin, Port Chester
E. I. Harrington, Yonkers

Officers. (May, 1903.)

Samuel Pashley, *President.* R. A. Heenan, *Vice-President.*
Henry Root, *Secretary and Treasurer.*

Censors: S. J. Banker, R. C. Davies, Lewis R. Oatman.
Committee on Hygiene: W. C. Cuthbert, J. W. Dean, William M. Rapp.

Members.

O. C. Baker, Brandon, Vt.
S. J. Banker, Fort Edward
Franklin T. Beattie, Shusan
F. H. Braymer, Knoxville, Tenn.
G. A. Bromley, Granville
Frank H. Carpenter, West Rupert
J. Cipperly, Middle Falls
James S. Cooley, Glen Cove
W. C. Cuthbert, Sandy Hill
R. C. Davies, Middle Granville
J. Wyman Dean, Sandy Hill
A. E. Falkenbury, Whitehall
C. J. Farley, Bolton
Fred. G. Fielding, Glens Falls
H. Gray, Greenwich
P. Z. Hebert, London, Eng.
Robert A. Heenan, Sandy Hill
B. R. Holcomb, Whitehall
S. B. Irwin, West Hebron
Edward Joslin, Whitehall
Charles W. Keefer, Mechanicsville
J. Knowlson, Poultney, Vt.
Charles E. Lambert, New York
J. Lambert, Salem
W. B. Madison, West Hebron
J. H. Maguire, Salem
D. M. McKenzie, Granville
Wm. B. Melick, Fort Edward
J. Millington, Greenwich
H. C. Monroe, Sandy Hill
I. T. Monroe, Granville
G. B. Murray, Greenwich
Lewis R. Oatman, Greenwich
James T. Park, Sandy Hill
Samuel Pashley, Hartford
William M. Rapp, Argyle
W. H. Renois, East St. Louis, Ill.
Henry Root, Whitehall
Sidney A. Rowe, Fort Ann
B. C. Senton, Rutland, Vt.
J. C. Sill, Argyle
George M. Stillman, Argyle
C. W. Sumner, North Granville
W. A. Tenney, Granville
T. C. Wallace, Cambridge
A. M. Young, Salem

Number of Members, 46.

MEDICAL SOCIETY OF THE COUNTY OF WAYNE.

(Organized July 2, 1823.)

MEETINGS.—Annual, second Tuesday in July, at Lyons; semi-annual, second Tuesday in January, at Newark.

Officers.

E. Hoag Lapp, *President.* G. D. Barrett, *Vice-President.*
George S. Allen, *Secretary.* Darwin Colvin, *Treasurer.*

Censors: M. A. Veeder, H. L. Chase, W. J. Coppernall, E. H. Lapp.

Members.

George Allen, Clyde
George S. Allen, Clyde
G. D. Barrett, Clyde
J. S. Brandt, Ontario
Mary A. Brownell, Newark
J. S. Campbell, Ontario
M. E. Carmer, Lyons
Robert S. Carr, Williamson
H. L. Chase, Palmyra
Darwin Colvin, Clyde
W. J. Coppernall, Newark
George A. Craft, Newark
L. B. Darling, Palmyra
Gard W. Foster, Clyde
Cyrus Fulton, Lyons
T. H. Hallett, Clyde
W. J. Hennessy, Palmyra
D. B. Horton, Red Creek
C. Jennings, Macedon
W. H. Jessup, Newark
N. E. Landon, Newark
F. A. Lewis, Walworth
E. H. Lapp, Williamson
J. F. Myers, Sodus
M. W. T. Negus, South Sodus
W. F. Nutten, Newark
James W. Putnam, Lyons
R. A. Reeves, Macedon
W. H. Richardson, Sodus
J. N. Robertson, Wolcott
H. F. Seaman, Alton
A. F. Sheldon, Lyons

L. H. Smith, Palmyra
Jennie M. Turner, Lyons
M. A. Veeder, Lyons
F. L. Wilson, Sodus Point
G. D. Winchell, Rose
George D. York, Newark
A. A. Young, Newark
Number of Members, 38.

Deceased: J. N. Arnold, Clyde, May 17, 1902, æt. 65; Timothy S. Fish, Wolcott, August 9, 1901, æt. 55.

MEDICAL SOCIETY OF THE COUNTY OF WESTCHESTER.
(Organized May 8, 1797.)

MEETINGS.—Annual, third Tuesday in May, at White Plains; stated, third Tuesday in September, November, January, and March. (Time and place of stated meetings at the option of the Society.)

Officers. (May, 1903.)

R. T. Irvine, *President.* E. I. Harrington, *Vice-President.*
D. M. Orleman, *Secretary.* S. O. Myers, *Treasurer.*
N. F. Curtis, *Curator.*

Censors: H. E. Schmidt, *Chairman,* W. S. Fleming, A. M. Campbell.

Committee on Hygiene: J. T. Gibson, *Chairman,* J. L. Porteous, C. N. Raymond.

Committee on Tuberculosis: T. F. Goodwin, *Chairman,* H. B. Brown, F. W. Shipman, E. F. Brush.

Committee on Legislation: G. C. Weiss, *Chairman,* W. D. Granger, W. F. Greene, R. T. Irvine, C. A. Knight.

Members.

S. W. Adams, Mt. Vernon
Oliver L. Austin, Tuckahoe
G. B. Balch, Yonkers
Lester C. Baldwin, Bedford
Garrett N. Banker, Yonkers
A. T. Banning, Mt. Vernon
M. W. Barnum, Ossining
P. J. Bauerberg, Yonkers
E. B. Beaudry, New Rochelle
W. A. Bell, Yonkers
A. C. Benedict, Yonkers
C. S. Benedict, 310 W. 102d st, N. Y.
James C. Bennett, Yonkers
T. P. Berens, New Rochelle
Samuel Beyea, New Rochelle
J. F. J. Blace, White Plains
T. E. Bleick, New Rochelle
J. W. Bowden, Yonkers
E. N. Brandt, Hastings-on-Hudson
H. Beattie Brown, Yonkers
Valentine Browne, Yonkers
E. F. Brush, Mt. Vernon
W. E. Bullard, Larchmont
Clarence W. Buckmaster, Yonkers
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